

copyright 2015 by Dr. M. Allam

All rights are reserved. No part of this book may be used or reproduced in any manner whatsoever without written permission, except in the case of brief quotations embodied in critical articles or reviews.

VISIT...

LANZAROTE
Caliente.COM

Dedication

Allah the all merciful, I beg thee to accept this effort for the soul of my father
He was your gift for me

Index

Subject	Page
Introduction	1
Cardiology scheme	3
symptomatology	4
Heart failure	10
Ischemic heart disease	25
Systemic Hypertension	48
Pulmonary hypertension	60
Rheumatic fever	62
Endocarditis	67
Pericardial disease	73
Myocarditis	83
Heart sounds	84
Valvular heart diseases	85
Murmurs	102
Cardiomyopathy	104
Dysarrhythmias	108
Aortic aneurysm and dissection	134
Cardiogenic shock	137
Sudden cardiac death	138

Introduction

Heart rate : (HR)

- It is the number of heart beats per minutes.
- Normally : 60 - 100 / min.
- It is controlled by autonomic nervous system:
 - ✓ Sympathetic stimulation increase the heart rate.
 - ✓ Parasympathetic stimulation decrease the heart rate.
 - ✓ No parasympathetic nerves supply the ventricles.

Stroke volume : (SV)

- It is the volume of blood pumped by each ventricle in one beat.
- Normally : 70 ml in the average (40 - 70 ml / m²).
- It depends on :
 1. Preload (volume load or diastolic load)..
 2. Afterload (pressure load or systolic load).
 3. Contractility (inotropic state).

Cardiac output : (CO)

- It is the volume of blood pumped by each ventricle in one minute.
- $CO = HR \times SV$.
- Normally : 5 liter / minute in the average (2.5 - 4.2 L / m² / min.) .
- It depends on :
 - ✓ Heart rate.
 - ✓ Preload.
 - ✓ Afterload.
 - ✓ Contractility.

Preload

- It is the degree of stretch of myocardium prior to contraction.
- Within limits, the stroke volume is directly related to preload.
- Starling's law: within limits, the greater the length of myocardial fibers the more powerful the contraction.

Afterload

- It is the resistance facing the ventricle during contraction.
- The stroke volume is inversely related to afterload.

Contractility

- It is the force of contraction of cardiac muscle.
- The stroke volume is directly related to contractility.

Ejection fraction : (EF)

- Ejection fraction is a test that determines how well your heart pumps with each beat.

Left ventricular ejection fraction (LVEF)

is the measurement of how much blood is being pumped out of the left ventricle of the heart (the main pumping chamber) with each contraction.

Right ventricular ejection fraction (RVEF)

is the measurement of how much blood is being pumped out of the right side of the heart to the lungs for oxygen.

In most cases, the term “ejection fraction” refers to left ventricular ejection fraction.

What do the numbers mean?

Ejection fraction is usually expressed as a percentage.

A normal heart pumps a little more than half the heart's blood volume with each beat.

A normal LVEF ranges from 55-70%.

A LVEF of 65, for example, means that 65% of the total amount of blood in the left ventricle is pumped out with each heartbeat.

The LVEF may be lower when the heart muscle has become damaged due to a heart attack, heart muscle disease (cardiomyopathy), or other causes.

An EF of less than 40% may confirm a diagnosis of heart failure.

Someone with diastolic failure can have a normal EF.

An EF of less than 35% increases the risk of life- threatening irregular heartbeats that can cause sudden cardiac arrest (loss of heart function) and sudden cardiac death.

An implantable cardioverter defibrillator (ICD) may be recommended for these patients.

Cardiology scheme

- Definition
- Etiology
- Clinical pictures :
 - ✓ Symptoms
 - ✓ Signs
 - ✓ Complications
- Investigations :
 - ✓ X ray
 - ✓ ECG
 - ✓ ECHO
 - ✓ Catheterization
- Treatment :
 - ✓ General measures
 - ✓ Treatment of the cause
 - ✓ Treatment of the precipitating factors
 - ✓ Treatment of the complications

Symptomatology

Dyspnea

Definition :

Uncomfortable awareness of breathing or Shortness of breath

Pathogenesis of Cardiac Dyspnea:

A. **Mechanical factors**

1. Pulmonary congestion :
 - Interstitial pulmonary oedema which leads to diminished alveolar compliance (the most important factor).
 - Intra-alveolar oedema.
 - Oedema of bronchial mucosa & bronchospasm.
2. Low cardiac output :
 - Leading to weakness of respiratory muscles.
3. Hydrothorax :
 - Leading to mechanical compression of the lungs.
4. Infradiaphragmatic causes :
 - Right-sided heart failure, pericardial effusion and constrictive pericarditis lead to ascites and enlarged tender liver which may elevate the diaphragm and decrease its mobility.
5. Massive pericardial effusion and huge cardiomegaly :
 - Occasionally causes mechanical compression of the lungs and bronchi.

B. **Nervous factors:**

Activation of Hering-Breuer reflex

- This reflex is present in normal individuals.
- In this reflex, impulses arise from stretch receptors in the terminal airways at the end of respiration. This leads to reflex inhibition of inspiration and passive relaxation of the chest .
- In left-sided heart failure, interstitial oedema activates this reflex leading to shallow rapid respiration.

Churchill–Cope reflex

- This reflex is not present in normal individuals.
- It occurs in pulmonary congestion in which distention of the pulmonary vessels stimulate J-receptors resulting in reflex stimulation of respiratory center causing tachypnea.

C. **Chemical factors:**

- Hypoxia occurs due to low CO & pulmonary congestion.
- Hypercapnia + Acidosis occurs due to tissue hypoxia.

Types of Cardiac Dyspnea:**A. Exertional dyspnea:** NYHA (New York Heart Association)

- * Grade I: dyspnea on more than ordinary effort (severe exertion).
- * Grade II: dyspnea on ordinary effort (moderate exertion).
- * Grade III: dyspnea on less than ordinary effort (mild exertion)
- * Grade IV : dyspnea at rest.

B. Orthopnea: Dyspnea on lying flat which relieved by sitting.

Pathogenesis:

- 1- Increased **venous return** aggravates pulmonary congestion.
- 2- Elevation of the **diaphragm**. (↓ vertical diameter)
- 3- Interference with the action of **respiratory muscles**.

C. Paroxysmal Nocturnal Dyspnea (PND)**Clinical picture:**

The patient arises **1-2 hours** after sleep with severe **inspiratory dyspnea**, cough ± frothy expectoration. _ wheeze maybe present (bronchial oedema & bronchospasm).

The condition is relieved **spontaneously** or with treatment - but may be recurrent.

Pathogenesis (1-3 as Orthopnea)

1. Increased venous return aggravates pulmonary congestion.
 2. Elevation of the diaphragm.
 3. Interference with the action of respiratory muscles.
 4. Absorption of oedema fluid into the circulation (VR).
 5. Decreased sympathetic activity during sleep (reduction of contractility).
 6. Nightmares: may lead to tachycardia & elevation of BP (load).
 7. Slipping down from high pillows.
- } as Orthopnea

- Orthopnea is not specific to cardiac diseases
- PND is a specific symptom of left sided heart lesion especially LSHF

* We have to exclude B.A.

Differentiation between Cardiac & Bronchial Asthma

	Cardiogenic	Bronchial
Age	Any age	Usually young age
Past history	Cardiac symptoms	Chest symptoms
Duration	Usually short	Usually long
Time of attack	1-2 hours after sleep	Early in the morning
Dyspnea	Mainly inspiratory	Mainly expiratory
Sputum	Frothy & may be bloody	Thick pellets
Chest exam.	Basal crepitations	Wheezes
Heart exam	Gallop & murmurs	Normal
ECG	Abnormal	Normal
Effect of drugs		
Adrenaline	Contraindicated	Improves the condition
Morphine	Improves the condition	Contraindicated
Aminophyllin	Improves the condition	Improves the condition

Oedema

Definition

Accumulation of excessive fluid in the interstitial spaces.

Pathogenesis of oedema

1. Increased capillary hydrostatic pressure:

- Increased venous pressure: HF, Pericardial effusion, constrictive Pericarditis, Venous occlusion e.g. thrombosis.
- Salt & water retention.

2. Decreased plasma osmotic pressure : (Due to hypoproteinaemia)

- Malnutrition.
- Malabsorption.
- Liver failure.
- Nephritic syndrome.

3. Increased capillary permeability:

- Inflammation: e.g. Infection, Allergy, Chemicals or Trauma.
- Hypoxia

4. Lymphatic obstruction

Generalized Oedema:

- **Cardiac:** Right-sided heart failure, pericardial effusion & constrictive pericarditis.
- **Hepatic:** Liver failure.
- **Renal:** Nephrotic syndrome & Nephritic syndrome.
- **Nutritional:** Malnutrition, Malabsorption.
- **Severe allergy.**

Pathogenesis of Cardiac Oedema:

A. Increased capillary hydrostatic pressure:

1. Increased venous pressure: due to stagnation of blood in the systemic veins.
2. Salt & water retention (see later)

B. Hypo-albuminaemia:

1. Diminished protein **intake** due to anorexia & vomiting.
2. Diminished protein **absorption** due to intestinal congestion.
3. Diminished albumin **synthesis** in the liver.
4. Increased protein **loss** in urine.

C. Increased capillary permeability: due to hypoxia.

Salt & water retention

1. Decrease Glomerular filtration rate & urine output : (Low cardiac output).
2. Secondary hyperaldosteronism:-
 - Secondary to low CO (reduction of renal blood flow ----- rennin)
 - Diminished destruction -----congested liver.
3. Increased ADH : due to :
 - Increased production secondary to low CO - Diminished destruction.
4. Decreased secretion of renal prostaglandins.

Features of Cardiac Oedema:-

1. **Dependent** : Oedema of lower limbs (Sacral oedema: bed-ridden patients).
2. **Bilateral & equal**: however, may be more in one side due to DVT, Postural (lye on one side).
3. **Lower limbs precedes ascites** : however, ascites may be the first " Ascites precox".
4. **Pitting & not tender**.
5. **Associated** with other features of cardiac disease.

Ascites precox

- a. Pericardial effusion & constrictive pericarditis: due to **narrowing** of hepatic veins --- marked liver congestion----- **obstruction of lymphatics**.
- b. TR & TS: because regurgitation of blood causes marked liver congestion.

Causes of Local Oedema

1. Venous obstruction.
2. Lymphatic obstruction.
3. Inflammatory oedema.

Syncope**Definition :**

Transient loss of consciousness due to acute cerebral ischaemia. If prolonged, convulsions may occur. Further prolongation causes coma, brain damage & death.

Pathophysiology :

Decreased cerebral perfusion is the final common pathway leading to syncope.

Clinical pictures :

Patients may describe a syncopal episode in many ways, including blackout, dizzy spell, and seizure or unexplained falls, particularly in elderly persons.

Aetiology1. Vasomotor syncope (**the commonest**)

A- Vasovagal syncope (neurogenic syncope): (known as **simple fainting**)

- Severe vagal stimulation --- severe bradycardia, hypotension, pallor & sweating.
- **Causes**: sudden severe fear, pain, and trauma (e.g. to testicles).

B- Carotid sinus syndrome:

Pressure on hypersensitive carotid sinus baroreceptors: e.g. during shaving.

2. Cardiac syncope (**Any cause of low CO**) especially:

- **A**ortic stenosis (or any other valvular obstruction).
- **A**cute heart failure (e.g. AMI).
- **A**rrhythmias (whether tachy- or brady-arrhythmia).
- **A**dams-stokes attacks.

- Usually exertional.
- No convulsions.

3. Cerebral syncope (**Reduced cerebral blood flow**)

- Vertebrobasilar TIAs.

4. Hypoxic syncope (**↓ O₂ content of the cerebral blood flow**)

- Fallot's tetralogy & other cyanotic diseases or severe anemia.

5. Postural syncope (**Orthostatic syncope**)

During standing:- sympathetic ---- reflex VC of BVs of L.Ls ----- prevent pooling of blood in lower limbs. failure of this mechanism leads to postural hypotension

Causes:

Reduce Sympathetic	- Autonomic neuropathy, e.g. Diabetes. - Sympatholytic drugs, e.g. ganglion blockers & vasodilators. - Lumbar sympathectomy.
Reduced Volume	- Hyponatremia - Hypovolaemia (Haemorrhage or dehydration) - Huge varicose veins.
Weak LL Ms	- Prolonged recumbency - Elderly patient. - Weakness of the muscles of the lower limbs (muscle pump).

6. Situational syncope

- Cough syncope (*Tussive syncope*).
- Micturition syncope (*more common in old men especially at night*).
- Defecation syncope.
explained as:- Straining → decreased VR → decreased CO → syncope.

**Cyanosis / palpitation / Jaundice and Fever in cardiac patient:-
see clinical book**

Haemoptysis in cardiac patients

Causes	Features
– Acute pulmonary edema. – Pulmonary infarction. – Pulmonary infection. – Pulmonary apoplexy. – Severe hypertension. – Ruptured aortic aneurysm into a bronchus. – Associated conditions causing haemoptysis	– Frothy blood-tinged sputum. – Frank haemoptysis. – Blood-streaked mucopurulent. – Profuse frank haemoptysis (rare).

N.B.

- It may occur due to rupture of bronchial varices occurring in chronic mitral stenosis.

Cardiovascular causes of chest pain

Origin	Causes
Myocardium	IHDs: angina - MI.
Pericardium	Pericarditis - pericardial eff.
Endocardium	Mitral valve prolapse.
Aorta	Aortic aneurysm - dissection.
Pulmonary	P. embolism.
Huge cardiomegaly:- Rare	

Cardiac neurosis:

- Neurotic patients especially females.
- Pain:- Localized left inframamary.
- Stitching, of variable duration. No relation to exertion. -Not relieved by rest.
- Associations other neurosis.
- Normal cardiac examination.

Don't forget

- **Analysis :** as any pain (11 points with stress on the 3 variants)

8 as usual

- ✓ Onset - Course - Duration.
- ✓ Association.
- ✓ What ↑ and what ↓.
- ✓ Effect of treatment.
- ✓ Date of last attack.

+ 3

- ✓ Site.
- ✓ Radiation.
- ✓ Character

Heart Failure

Definition

Inability of the heart to **maintain cardiac output** sufficient to metabolic **requirements** of the body *in spite of adequate venous return*.

Classification of HF

1. Left-sided heart failure, right-sided heart failure or biventricular.
2. Acute & chronic heart failure.
3. Systolic & diastolic failure.
4. Aetiological classification e.g. Ischaemic heart failure.
5. According to cardiac output:

Low C.O.P. failure	High C.O.P failure
- C.O.P. less than 5L/min - most of HF cases except those with hyperdynamic circulation	- C.O.P. high but net insufficient for tissue needs. - This occurs in hyperdynamic circulation - Treated by treatment of the cause.

Aetiology of heart failure

Left Atrium		Right Atrium	
	* M.S * Left atrial myxoma		* Pulmonary hypertension - T.S . * Right atrial myxoma
Left ventricle		Right ventricle	
Increased Volume overload	* Mitral incompetence - Aortic incompetence. * VSD & PDA. * Hyperdynamic circulation.	* Tricuspid incompetence. * VSD & PDA. * Hyperdynamic circulation.	
Increased Tension overload	* Systemic hypertension (HTN). * Aortic stenosis. * Coarctation of aorta.	* Pulmonary hypertension. : Mitral stenosis. : LVF. : Chest dis. (Cor-pulmonale). * Massive pulmonary embolism. * Pulmonary stenosis.	
Decreased contractility (Myocardial disease)	* Ischemic heart disease especially MI. * Cardiomyopathy. * Myocarditis.		

Causes of biventricular failure:

- * + right ventricular failure on top of left ventricular failure (**congestive HF**).

Pathophysiology

➤ **Compensatory mechanisms:**

The presence of underlying heart disease leads to compensatory mechanisms which is able to prevent the appearance of manifestations of heart failure under basal conditions (compensated heart failure). These mechanisms include:

1. Ventricular dilatation: this occurs in cases with increased preload, and according to Starling law, this dilatation will lead to increase in CO. However, if the dilatation exceeds certain limit, the ventricular contractility will decline.

2. Ventricular hypertrophy: this occurs in cases with increased afterload. The increase in muscle mass will lead to increase in cardiac contractility and CO. However, since this hypertrophy is not associated with increase in blood supply, ischemia of the hypertrophic muscle fibers will lead to fibrosis and decline in cardiac contractility by time.

3. Tachycardia: Since $CO = SV \times HR$, reduction in SV due to cardiac disease is compensated for by increase in HR to maintain CO. The increase in HR occurs through the following mechanism:

- Marry's law: HR is inversely proportionate to blood pressure.
- Bainbridge reflex: Increased RA pressure causes increased HR
- Marked tachycardia may lead to diminished CO due to decreased diastolic (filling) time.

4. Redistribution of blood flow: Activation of sympathetic nervous system due to low CO will lead to diversion of blood flow from less vital organs (e.g. skin) to more vital organs (e.g. brain & heart).

5. Hypervolemia:

- Reduction in CO will lead to salt and water retention through:
 - ✓ Reduction of GFR.
 - ✓ Renal ischemia $\rightarrow \uparrow$ renin $\rightarrow \uparrow$ Ang II $\rightarrow \uparrow$ aldosterone $\rightarrow$ salt & water retention.
 - ✓ Increased ADH $\rightarrow$ water retention.
- Increased blood volume will increase preload and cardiac contraction.
- If it exceeds certain limits, it will reduce cardiac contractility and lead to edema.

If the compensatory mechanisms exceed their limits, or if the cardiac load or damage is increased, manifestations of heart failure will appear (decompensated heart failure).

➤ **Neuro-hormonal changes:**

Reduction of CO will lead to activation of neuro-hormonal mechanisms aiming at maintaining perfusion pressure to body organs, however this may be on the expense of increasing cardiac load and requirements. These mechanisms include:

1. Activation of renin-Angiotensin- aldosterone system.

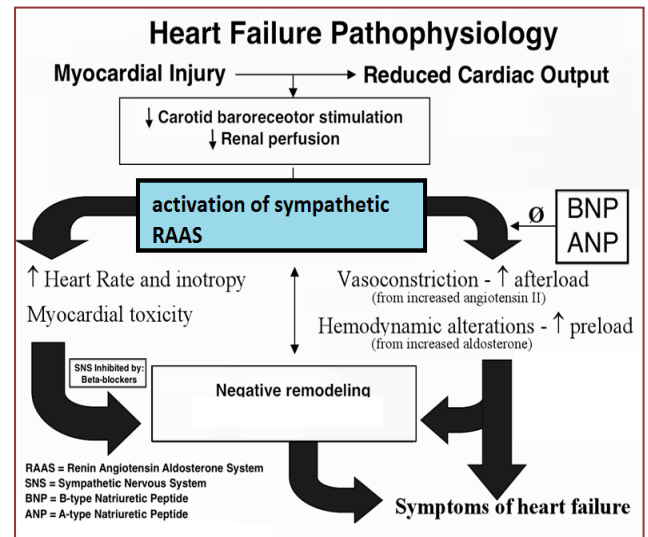
2. Increased catecholamine level: in chronic heart failure there is:

- Reduction of adrenergic receptor level (down regulation).
- Reduction of G-protein activity that couples α -receptors to adenylyl cyclase system.
- Reduction of c-AMP $\rightarrow \downarrow$ intracellular Ca $\rightarrow$ reduce contractility.

➤ **Precipitating factor:**

These factors may cause cardiac decompensation due to overwhelming of the cardiac compensatory mechanisms. These include:

- **I**nfections: as infective endocarditis, Lung infection.
- Myocardial **i**nfarction.
- **I**atrogenic: discontinuation of anti failure therapy excess salt, IV fluids, steroid, -Ve inotropic (B blocker)
- **P**hysical and emotional stress.
- **P**regnancy & labour.
- **P**ulmonary embolism.
- **A**rrhythmia e.g. marked tachycardia or bradycardia.
- **A**nemia, thyrotoxicosis,,, hyperdynamic circulation.



Clinical pictures

Left-sided heart failure

- 1- forward:- Low COP
- 2- Backward:- P. Congestion
- 3- CVS:- General + Local

A) Forward manifestations (low COP) "after"

Symptoms	Signs
– Easy fatigability & LL intermittent claudication. – Dizziness, blurring of vision & may be syncope. – Anginal pain in severe cases. – Oliguria in severe cases	– Pallor, coldness & peripheral cyanosis. – Weak pulse. – Low systolic pressure.

B) Backward manifestations (pulmonary congestion) "before"

Symptoms	Signs
– Dyspnea: – Exertional dyspnea --- dyspnea at rest – Orthopnea – Paroxysmal nocturnal dyspnea ---- PND. – Cough - Hemoptysis - Recurrent chest infections	– Bilateral basal crepitations (BBC). – Pleural effusion (hydrothorax). – In severe cases: acute pulmonary edema (APO)

C) Cardiovascular signs

1. General signs:

- **Tachycardia**: due to sympathetic stimulation
but may be absent:- in cases associated with HB, digitalis, B. Blockers.
- **Weak pulse**.
- **Pulsus alternans**: alternating strong & weak beat with equidistance.
- Systolic pressure: **low**

2. Local signs:**Precordial examination**

- Signs of left ventricular enlargement.

Auscultation

- Ventricular (protodiastolic) gallop "at apex".
- Murmur of functional mitral incompetence due to left ventricular dilatation.
- Features of the cause may be detected.

Right-sided heart failure

- 1- forward:- Low COP
- 2- Backward:- S. Congestion
- 3- CVS:- General + Local

A) Forward manifestations (low COP) "after" See above.

B) Backward manifestations (systemic congestion) "before"

Symptoms

- Pain in the right hypochondrium & epigastrium.
- Dyspepsia, anorexia, nausea, vomiting & may be malabsorption in severe cases.

Signs

- Enlarged tender liver & may be cardiac cirrhosis in chronic failure cases.
- Congested pulsating neck veins.
- Ascites. - Pleural effusion (hydrothorax).
- Oedema of the lower limbs.

C) Cardiovascular signs**1. General signs:**

- **Tachycardia:** due to sympathetic stimulation
but may be absent:- in cases associated with HB, digitalis, B. Blockers.
- **Weak pulse.**
- **Systolic pressure: low**

2. Local signs:**Precordial examination**

- Signs of Right ventricular enlargement.

Auscultation

- Ventricular (protodiastolic) gallop "at Tricuspid area".
- Murmur of functional Tricuspid incompetence due to Right ventricular dilatation.
- Features of the cause may be detected.

American Heart Association Stages of Heart Failure

Stage	Description
A: High risk for developing HF	Hypertension, diabetes mellitus, CAD, +Ve family history
B: Asymptomatic HF	Previous disorders:- MI, LV dysfunction, valvular heart disease
C: Symptomatic HF	Structural heart disease - SOB - fatigue - impaired exercise tolerance
D: Refractory end-stage HF	Marked symptoms at rest despite maximal medical therapy

Summary

	LVF	RVF
Forward manifestations	Low COP	
	Symptoms • Syncope. • anginal pain and oliguria. • Intermittent claudication.	Signs • Pallor, coldness & peripheral cyanosis. • Weak pulse. • Low systolic pressure.
Backward manifestations	Pulmonary congestion Symptoms • Dyspnea, orthopnea & PND. • Cough. • Haemoptysis. • Recurrent chest infection. Signs • Bilateral basal crepitations (BBC). • Pleural effusion. • APO.	Systemic congestion Symptoms • Right hypochondrial pain. • Dyspepsia. • L.L. edema. Signs • Enlarged tender liver. • Congested neck veins. • Ascites. • Pleural effusion. • L.L. edema.
General signs	• Tachycardia "except???" • Weak pulse. • Pulsus alternans. • Low systolic pressure. • Pleural effusion (hydrothorax).	• Tachycardia "except?" • Weak pulse. • Low systolic pressure. • Pleural effusion (hydrothorax).
Local cardiovascular signs	• Signs of LV enlargement. Auscultation • S3 gallop (at apex). • Murmur of functional MR due to LV dilatation. Features of the cause	• Signs of RV enlargement. Auscultation • S3 gallop (at tricuspid area). • Murmur of functional TR due to RV dilatation. Features of the cause

Investigations

A. X-ray	• Chamber enlargement. • Pulmonary vascular (left sided failure). • Pleural effusion (Right or left HF). • Calcified valve.
B. ECG	• Chamber enlargement (mainly hypertrophy). • Aetiology (ischaemic)
C. Echo & Doppler	• Cause (e.g. diagnosis of valve lesion or congenital defects). • Site. • Effect (chamber enlargement). • Function (ejection fraction).
D. Cardiac Catheter	• As Echo and doppler

N.B.

- **Transesophageal echocardiography** : is particularly useful in patients who are on mechanical ventilation or morbidly obese and in patients whose transthoracic echo was suboptimal.
- **B-type natriuretic peptide (V. important) Diagnostic BNP**: amino acids polypeptide originated from the ventricles.
- **Radionuclide multiple gated scan**: Very reliable imaging technique for determining global heart function.
- **CBC, KFTs and electrolytes**: Mild anemia can worsen prognosis.

Treatment of heart failure**Guidelines for treatment of heart failure**

1. **Treatment of the underlying etiology.**
2. **Treatment of the precipitating factors.**
3. **Treatment specific to the syndrome of HF:**
 - **Decreasing the cardiac load:**
 - a. Rest: physical and emotional.
 - b. Reduction of preload:
 - Diet control, especially salt restriction.
 - Diuretics.
 - Vasodilators.
 - c. Reduction of after load:
 - Vasodilators.
 - **Decreasing the myocardial damage:**
 - Beta blockers.
 - **Increasing the myocardial contractility:**
 - a. Digitalis.
 - b. Other inotropic agents:
 - Sympathomimetic **amines**: dop**amine** & dobut**amine**.
 - Phosphodiesterase inhibitors: am**rinone** & mil**rinone**.
 - **Resynchronizing the ventricles: "CRT"**
 - In patients with cardiomyopathy who have the problem of :
Intraventricular conduction delay or bundle branch block
 - **Symptomatic treatment:**
 - a. For hypoxemia: oxygen administration.
 - b. For cardiac dyspnea: aminophylline administration.
4. **Treatment of the refractory heart failure.**
5. **Treatment of acute pulmonary edema.**

1. Rest

- Reduce metabolic requirements

- **Semi-sitting or sitting position:** to decrease venous return & improve respiratory muscles.

Complications of prolonged recumbency:

Peripheral	Central	Locomotor system
- DVT.	- <i>P embolism.</i>	- <i>Muscle wasting.</i>
- Bed sores.	- <i>Hypostatic pneumonia.</i>	- <i>Osteoporosis.</i>
	- <i>Constipation.</i>	- <i>Joint stiffness.</i>
	- <i>Depression.</i>	

2. Sedative

To relieve anxiety (Diazepam or Morphine; the best of APO)

3. Diet

- Salt restriction:** To reduce salt & water retention decreasing blood volume (reduction of preload) (reduction of circulatory congestion & edema).
- Fluid restriction:** in severe & resistant cases of heart failure.
- The meals:** light of carbohydrate mainly and fractionated.
- Caloric restriction:-** Reduction of body weight in obese patients.

4. Diuretics

They promote loss of salt & water decreasing blood volume (reduction of preload).

Diuretics classified into

Diuretic	Site and mechanism	Side effects
Loop Diuretic • Frusemide (lasix) 40-160 mg (O/IV) • Bumetanide • Ethacrynic acid: 50 – 200 mg (O/IV)	Thick ascending loop of Henle inhibition of Na- K & KCL co-transporter	Hypo: kalaemia, natraemia, calcaemia , dehydration, alkalosis. Hyper: glycemia, lipidemia, uricemia. Others: blood dyscrasia, rashes, ototoxicity, GITD, interstitial nephritis
Thiazide • Hydrochlorothiazide 25 – 100 mg /d Or • Chlorothalidone 25 – 100 mg /d Or • Indapamide *	DCT Inhibition of NaCl cotransport Vasodilator	Hypo: kalaemia, natraemia, dehydration, alkalosis Hyper: glycemia, lipidemia, uricaemia, calcaemia Others: Thrombocytopenia, rashes, pancreatitis, impotence.
K sparing • Spironolactone 50 – 200 mg • Triamterene 100 – 300 mg Amiloride 5 – 10 mg /d Or	Aldosterone antagonists Inhibition of Na conductance	Gynaecomastia + Hyperkalaemia Acidosis

*Works even if GFR is below 50 ml/min.

Other diuretics:

- Osmotic diuretics.
- Carbonic anhydrase inhibitors e.g. acetazolamide.
- Drugs with a diuretic action e.g. dopamine, digitalis.
- Mercurial diuretics (not use now).

Causes of diuretic resistance:

- Delayed absorption of the diuretic.
- Reduced secretion of the diuretic into the tubular lumen (its site of action).
- Compensatory retention of sodium after the effective period of the diuretic.
- Hypertrophy and hyperplasia of epithelial cells of the distal convoluted tubule.

5. Vasodilators

Aim reduction of preload (venodilatation) and after load (arteriolar dilatation).

Arteriolar	Venous	Both
Reduce afterload	Reduce preload	Reduce both
➤ Hydralazine. ➤ Diazoxide.	➤ Nitrates	➤ ACEIs. ➤ Na nitroprusside.

1. Angiotensin-converting enzyme inhibitors (ACEI): e.g.

- Captopril : 6.25 - 25mg (short duration of action → 8h orally).
- Enalapril - Lisinopril .(long duration of action → once daily).

N.B.

- ACEI are used in the treatment of H.F., hypertension, diabetic microalbuminuria and secondary hyperaldosteronism.
- Hypotension, hyperkalemia, dry irritating cough, metallic taste of mouth and renal impairment especially in cases with reduced renal blood flow → are side effects.

2. Ang II receptor Blockers (ARBs):

- Losartan
- Candesartan

ARBs:- Highly recommended alternatives to ACE inhibitors in patients who cannot tolerate ACE inhibitors because of adverse effects, most notably coughing.

3. Direct vasodilators: e.g.

- Nitroglycerine: 0.5 - 5 mg.
- Nitroprusside: 0.5 - 10 µg /kg/min IV Infusion.
- Alpha-blockers: e.g. Prazocin (mini press) → used in patients with renal impairment when ACEI cannot be used.

4. Calcium channel- blockers :e.g.

- Nifedipine.

They are rarely used in heart failure due to their negative inotropic effect.

5. Hydralazine: was the first oral balanced (afterload and preload reduction) vasodilator and was popular before the availability of ACE inhibitors.

6. Beta-adrenergic blocking agents (metoprolol, carvedilol)

- Improve symptoms and exercise tolerance,
- Reduce ABP so improve LV ejection fraction (**decrease mortality rates**) (ischemic and idiopathic cardiomyopathy).

Was not used before

6. Inotropic Agents

1. Digitalis glycosides.
2. Sympathomimetics (Catecholamines) :
 - **Dopamine:** 3-10 ug /kg /min IV infusion
 - **Dobutamin:** 5-10 ug /kg /min IV infusion
3. Phosphodiesterase inhibitors : Amrinone, milrinone.
4. Glucagon.
5. Calcium.

Digitalis

➔ **Mechanism of action:**

- Inhibition of membrane Na-K ATPase pump: This will increase intracellular Na → increase intracellular Calcium with Na or released from sarcoplasmic reticulum → stimulation of sliding of actin over myosin with increase in cardiac contractility
- Vagal stimulation
- Direct inhibition of A-V conduction
- Increase automaticity of Hiss-Burkinjie system and excitability of atrium and ventricular myocardium
- Direct diuretic effect(weak) .

➡ **Action :**

1. Increases myocardial **contractility** (i.e. positive inotropic effect) leading to :
 - Increased SV & CO. – Increased systolic BP.
 - Increased renal blood flow & urine output.
 - Decreased venous pressure. – Decreased size of the heart.
2. Increases **excitability** of atria & ventricles (may be complicated by arrhythmias).
3. Decreases the **automaticity** of SAN (direct & vagal++) slowing the rapid heart.
4. Decreases the **conductivity** of AVN (may be complicated by heart block).
5. Vagal stimulation.
6. ECC changes
 - Digitalis effect: sagging depression of ST segment.
 - Flat or inverted T wave.

➡ **Digitalis toxicity :**

different types of arrhythmias & heart block may occur.

➔ **Indication:**

- Heart failure.
- Atrial fibrillation, atrial flutter & paroxysmal supra ventricular tachycardia.

➔ **Contraindications:**

Absolute	Relative
- Digitalis toxicity. - Ventricular tachycardia (++ excitability)	- Partial Heart block (decrease conductivity). - Nodal rhythm (suppression of S-A node).

➡ **Preparations :**

- **Oral:** (one tablet equals one international unit)
 - Digoxin: excreted mainly by the kidney (tab = 0.25mg).
 - Digitoxin: metabolized mainly by the liver (tab = -0.1 mg).
- **Intravenous preparations:**
 - Digoxin (amp. Contains 0.5 mg).
 - Cedilanid.
 - Ouabin.

➔ **Administration :**

- **Oral:- Previously**

A loading or digitalizing dose of 12-15 units over:

- **One day (rapid digitalization)**
- **3 Days (moderate digitalization)**
- **7 Days (slow digitalization)**

This is followed by a maintenance dose of 1-3 units/day.

OR *It is given as follows:*

- **A dose of 2 units daily without loading.**
- **An optimum therapeutic level is reached after about 5 days.**
- **This is followed by a maintenance dose of 0.5 - 2 units/day.**

✓ **Now:** Dose of 0.5 - 1 units given daily with stoppage of 1-2 days weekly.

- **Intravenous**

Indications: Acute HF, AF, A. flutter & PSVT.

Dose: 0.5 mg / 4h (0.5-1 mg in 5% glucose over 30 min) (not exceed 1.5 mg)

Then maintenance oral dose of 0.5-2 nits/day (not exceed 2.5 units/d)

➡ **Digitalis drugs interaction :**

Amiodarone , propafenone, quinidine, verapamil, nifedipine, diltiazem, levothyroxine, cyclosporine, flecainide, disopyramide, omeprazole, tetracycline, and erythromycin.

Digitalis toxicity

Predisposing factors:

- Hypokalemia.
- Hypercalcemia
- Renal failure.
- Liver failure

Clinical picture:

- GIT: Anorexia (the first symptom) - nausea & vomiting.
- CVS: Different types of arrhythmias & heart block
 - Extrasystole (pulsus bigeminus or trigeminus)
 - Ventricular tachycardia.
- CNS: Blurring of vision & colored vision (yellow or green).
 - Headache, psychosis & neuralgia.
- With prolonged use → Gynaecomastia may occur.

Treatment :

1. **Stoppage** of digitalis.
2. Correction of **predisposing factors** :
 - Correction of hypokalaemia if present by:
 - Reduction of the dose or stoppage of loop diuretics
 - Potassium chloride orally with monitoring of serum K (by ECG):
 - Calcium-chelating agents e.g. EDTA are rarely used now.
3. **Symptomatic** treatment:
 - For arrhythmias: antiarrhythmic drugs e.g. propranolol & lidocaine.
 - For heart block: atropine IV.
 - For vomiting: antiemetic : Metoclopramide (Primperan) .
4. **Immunotherapy**: Digoxin immune Fab Immunoglobulin fragment with specific and high affinity for both digoxin and digitoxin molecules.
5. **Binding** agents: to reduce absorption e.g. Activated charcoal.
6. **Cholestyramine**: Used to break enterohepatic circulation.

Dopamine

(sympathomimetics agent) 3-10 ug / kg / min IV infusion = **dose dependent**.

Dose	Stimulate	Effect
Low (0.53 mcg / kg / min)	Renal vascular beds	Diuresis (Nephrogenic)
Moderate (3 – 10 mcg / kg / min)	Beta receptors	Inotropic (Heart)
High dosage (10 - 20 mcg / kg / min)	Alpha receptors	In shock (Shock)

Dobutamine

- (sympathomimetic agent) 5-10 ug / kg / min IV infusion.
- Beta 1-receptor agonist, with some beta 2- receptor and minimal alpha-receptor.
- Contraindicated in patients with moderate or severe hypotension.

Noradrenaline

- α -receptor effect mainly. Better avoided as it increases afterload by intense vasoconstriction

Adrenaline

- has both α and β -receptors effect. Used in refractory cases.

Phosphodiesterase inhibitors

- (milrinone, amrinone) (Oral PDIs have no role).
- Phosphodiesterase inhibitors (PDIs) increase intracellular cAMP, which results in a positive inotropic effect on the myocardium and peripheral vasodilation.

Don't forget

- Catecholamines stimulate Beta receptors $\rightarrow$ activation of Adenyl cyclase $\rightarrow$ $\uparrow$ CAMP $\rightarrow$ contractility.
- Phosphodiesterase $\rightarrow$ $\downarrow$ CAMP $\rightarrow$ $\downarrow$ contractility.

7. Aminophylline

Actions	- Bronchodilator. - Vasodilator. - Positive inotropic. - Mild diuretic
Indications	- Bronchial asthma. - Cardiac dyspnea
Administration	- Intravenous: 5.6 mg/kg slowly IV over 20 min. (average 250 – 500 mg). - Orally or per-rectum.
Side effects	- Hypotension. - Arrhythmias. - Insomnia. - Irritability.

8. Venesection:

- Closed or open venesection is rarely used in severe & resistant cases. (not now)

9. Dialysis:

- May be indicated in severe cases.

Treatment of diastolic HF

- A. Pericardiectomy** for constrictive pericarditis.
- B. Relief of ventricular systolic overload:** ACE inhibitors or ARBs.
- C. Anti-ischaemic agents:**
 - B- Blockers - Ca. Blockers - Nitroglycerin
 - (Reducing myocardial ischemia, thus improving ventricular relaxation).
- D. Treatment of the cause:** Relief of Valvular, Supravalvular, and subvalvular obstruction to ventricular out-flow by operation or balloon valvuloplasty.

Acute pulmonary edema

Definition

Rapid accumulation of fluid in interstitial & intra-alveolar spaces of the lung.

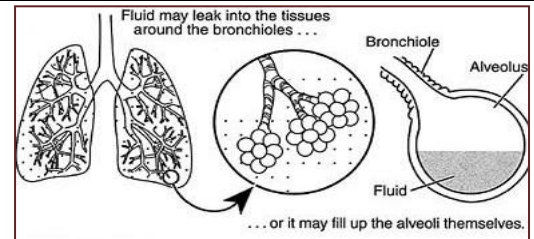
N.B.

- **Cardiogenic pulmonary edema (CPE)** is defined as development of pulmonary edema due to increased capillary hydrostatic pressure secondary to elevated pulmonary venous pressure (when LV venous return exceeds LV output).

Etiology

A. **Increased pulmonary capillary hydrostatic pressure: (cardiogenic pulmonary edema)**

- Acute left-sided heart failure.
- Acute myocardial infarction.
- Severe hypertension.
- Myocarditis.
- Acute mitral or aortic incompetence.
- **Acute exacerbation of chronic LSHF (P congestion)**: e.g. MS with precipitating factors as AF.



B. **Increased pulmonary capillary permeability: adult respiratory distress syndrome ARDS (Non-cardiogenic pulmonary edema)**

- | | |
|---|--|
| <ul style="list-style-type: none"> • Shock. • Trauma. • DIC. • Aspiration of gastric contents. • Pulmonary lymphatic obstruction. • Pancreatitis. • Inhalation of gases e.g. chlorine, 100 % oxygen. | <ul style="list-style-type: none"> • Septicemia. • Burns. • Severe pneumonia. • Severe hypoalbuminaemia. • High altitudes. • Terminal renal & liver failure. • C.N.S. lesions e.g. cerebrovascular strokes & head injuries. |
|---|--|
- Sudden expansion of a collapsed lung e.g. in rapid aspiration of massive pleural effusion.

Clinical pictures

- Severe dyspnea at rest & orthopnea.
- Cough with expectoration of excessive frothy blood-tinged sputum.
- Central cyanosis.
- Marked irritability, sweating & coldness.
- Bilateral crepitations, at first basal then generalized.
- Wheezes may be present.
- Features of the cause e.g. myocardial infarction.

Investigations

- **CBC**: severe anemia or sepsis.
- **Serum electrolytes**: hypokalemia and hypomagnesemia.
- **BUN and Creatinine**.
- **Chest X-ray films**:
 - ✓ Cardiomegaly.
 - ✓ Alveolar edema with pleural effusions (bilateral infiltrates in a butterfly pattern).
 - ✓ Haziness of hilar shadows & loss of sharp definition of pulmonary vasculature.
 - ✓ Thickening of interlobular septa (Kerley B lines).
- **Arterial blood gases**:
 - ✓ Stage 1 and 2: hypoxemia and hypocapnia.
 - ✓ Stage 3: hypoxemia, in severe cases hypercapnia.
- **Pulse oximetry**:
For monitoring the patient's response to supplemental oxygen and other therapies.
- **ECG**:
Not diagnostic.

Treatment of cardiogenic pulmonary oedema (APO)

1. Hospitalization: preferably in ICU & bed rest in sitting position.
2. Morphine: 5 – 10 mg IV.
3. Correction of the cause

Respiratory care	Cardiac care	Circulatory (hypervolaemia)
4. O2 inhalation.	6. Digoxin.	8. Frusemide.
5. Aspiration of secretion.	7. Others e.g. Dopamine & Dobutamine.	9. Vasodilators.
	10. Aminophylline	

If no response

11. Mechanical ventilation.
12. Intra-aortic balloon.
13. Phlebotomy or dialysis.

N.B.

- **Frusemide**: 20-40 mg IV, repeated every 30 min, according to the response.
- **Vasodilators**: Nitroglycerine IV or sub, or Na nitroprusside 0.5 - 10 ug / kg / min IV.
- **Aminophylline**: 5.6 mg/kg slowly IV over 20 min. (average dose : 250-500 mg)
- **Positive inotropics**: Digoxin may be used especially in cases associated with AF, Other inotropics may be needed e.g. Dopamine & Dobutamine.

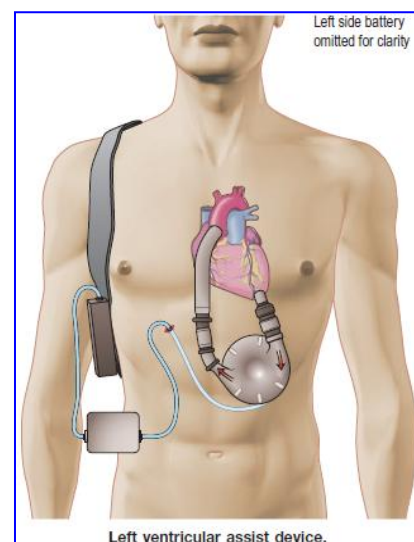
Refractory heart failure

Etiology of refractory heart failure

1. Presence of a mechanical factor hindering the cardiac function: e.g.
 - *Severe valvular disease.*
 - *Pericardial effusion or constrictive pericarditis.*
2. Persistence of the cause: e.g. uncontrolled hypertension.
3. Presence of a precipitating factor: e.g. chest infection.
4. Improper treatment: e.g.
 - *Inadequate rest.*
 - *Inadequate salt restriction.*
 - *Inadequate digitalis.*
5. Terminal cases of heart failure: with advanced myocardial damage, e.g. massive myocardial infarction.

Treatment

1. Removal of the **mechanical factor**: e.g. surgical treatment of valvular disease.
2. Treatment of the **cause**: e.g. proper control of hypertension.
3. Removal of the **precipitating** factor: e.g. proper antibiotics for chest infection.
4. Proper management:
 - *Adequate rest.*
 - *Adequate salt restriction (< 1 gm / day) & fluid restriction in severe cases.*
 - *Adequate digitalis.*
5. For terminal cases of heart failure:
 - *Diuretics*: use combinations e.g. loop diuretics & potassium-sparing diuretics.
 - *Vasodilators*: use combinations of IV vasodilators such as **Na nitroprusside** and a potent IV inotropics such as **Dopamine** or **Dobutamine**.
 - ***Mechanical measures***:
 - ✓ Mechanical removal of fluid using phlebotomy or dialysis.
 - ✓ Mechanical assistance of circulation using intra-aortic balloon counterpulsation: "**Balloon is inflated in diastole & deflated in systole**".
 - *Cardiac transplantation*: in patients below the age of 60.



Ischemic heart disease

Anatomy Of Coronary Arteries

The heart is supplied by two coronary arteries that originate from the root of the aorta and surround the heart as a crown (Coronary = crown).

	The left Coronary artery		Right Coronary
Origin	Left sinus of Valsalva		Right sinus of Valsalva
Site	Left atrioventricular groove		Right A-V groove
Branches	Anterior descending anterior inter-V groove → apex → turns backwards	The circumflex artery left A-V groove	Marginal a + posterior descending a
Anastomosis	posterior descending a	Right coronary	circumflex artery
Supply	Anterior surface	marginal branches Lateral surface	Inferior surface

Pattern of coronary supply

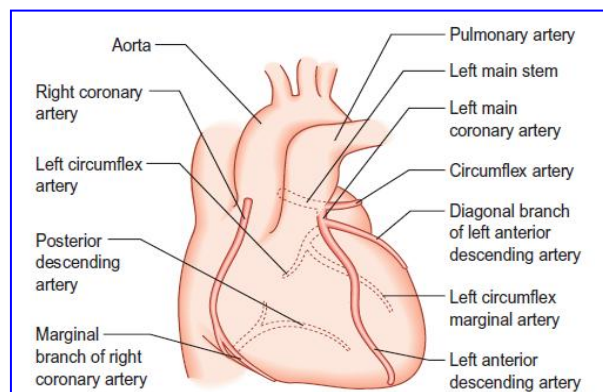
1- Balanced circulation:

Left coronary: supplies L atrium, L ventricle + *anterior part of the inter-V septum*.

Right coronary: supplies R atrium, R ventricle + *posterior part of the inter-V septum*.

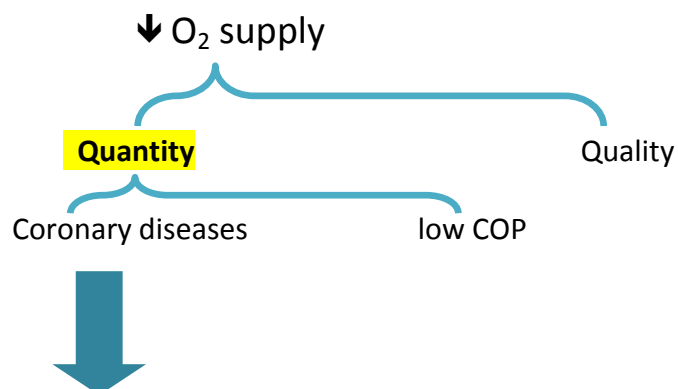
2- Right dominance: Right coronary supplies also the posterior part of the L. ventricle.

3- Left dominance: Left coronary supplies also the posterior part of the septum & the posterior wall of the right ventricle.



Aetiology of ischemic heart disease

Summarized as → O_2 demand > O_2 supply



↑ O_2 demand:

- 1- Tachycardia.
- 2- Increased contractility.
- 3- Increased after load (HTN & AS) = ventricular hypertrophy.
- 4- Increased preload (Hyper dynamic states)
- 5- severe stress (physical and mental).

Decreased oxygen supply

A. **Decreased quantity of coronary blood:**

1. **Coronary artery disease:** in ability of coronary to maintain sufficient blood flow in spite of good cardiac output.

Intima	• Atherosclerosis (the most common cause). • Vasculitis e.g. PAN
Osteum	• Coronary osteal stenosis e.g. syphilis, calcification. • Congenital anomalies.
Musculosa	• Dissection. • Spasm.
Lumen	• Embolism. • Coronary thrombosis in hyper-coagulability (polycythaemia).
Others	• Microcirculation vasoconstriction. • Spontaneous coronary dissection.

2. **Low cardiac output states:** e.g. valvular stenosis especially AS, Pulmonary hypertension.

B. **Decreased quality of coronary blood**

- Hypoxia.
- Severe anemia.

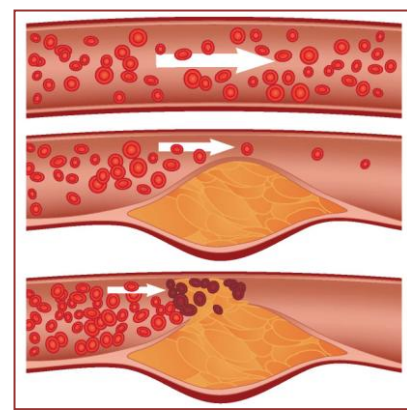
Risk factors of atherosclerosis

Non-modifiable

- Age → (Major)
 - Male sex → (Major)
 - Family history → (Major)
- Close relatives of complicated atherosclerosis (IHDs or stroke) + Genetic (as familial hypercholesterolemia) .*

Modifiable

- Systemic hypertension → (Major)
- Cigarette smoking → (Major)
- Obesity.



Partially Modifiable

- Diabetes Mellitus → (Major)
 - Hypercholesterolemia → (Major)
 - Dyslipidaemia → (Major)
- ✓ High LDL and / or VLDL
 - ✓ Low HDL "protective "
 - ✓ LDL/HDL ratio > 3:1

Ps

- Physical inactivity.
- Psychic stress.
- Personality (mentally active ambitious individuals).

Others

- Elevated serum levels of triglycerides + homocysteine + uric acid + fibrinogen + Elevated serum insulin levels (Insulin resistance).
- Elevated C-reactive protein.
- Vitamin B6 deficiency.
- Hypercoagulability.
- Postmenopausal estrogen deficiency.
- High intake of saturated fat + carbohydrate.

Metabolic syndrome:

1. DM:- hyperinsulinemia " insulin resistance"
2. Abdominal obesity
3. Decreased HDL cholesterol levels
4. Hypertension

Preventive factors

- A high serum HDL cholesterol level.
- Physical activity.
- Estrogen.
- Moderate alcohol intake (1 – 2 drinks /day). ☹

Presentation of ischemic heart disease "clinical pictures"

- A. **Asymptomatic**: silent myocardial ischemia.
- B. **Symptomatic**:
 1. Chest pain:
 - Angina pectoris: stable angina, unstable angina, variant angina.
 - Acute myocardial infarction.
 2. Other presentation: (with or without chest pain) e.g.
 - Heart failure.
 - Arrhythmias.
 - Fatigue.
 - Syncope.
 - Sudden death.

Angina pectoris

Definition

clinical term used to describe **chest pain** resulting from **brief** myocardial ischemia (oxygen demand > oxygen supply).

Pathophysiology

Chemical and mechanical stimulation of sensory afferent nerve endings in the coronary vessels and myocardium. These nerve fibers extend from the T1 – T4.

- **Chemical:** anaerobic metabolism of myocardium accumulates lactate & pyruvate. Adenosine may be the main chemical mediator of anginal pain.
- **Mechanical:** Heart rate, myocardial inotropic state and myocardial wall tension are the major determinants of myocardial metabolic activity and myocardial oxygen demand.

Aetiology

The same as ischemic heart disease, but the most common cause is → atherosclerosis.

- **Coronary atherosclerosis:**

This a descriptive term for hardening and thickening of the medium and large muscular and elastic arteries. It results from deposition of fat in the subintimal tissue with macrophage and fibroblast proliferation resulting in narrowing of the vessel lumen by itself, by the subintimal hemorrhage or by inducing clot formation over the damaged abnormal vascular endothelium.

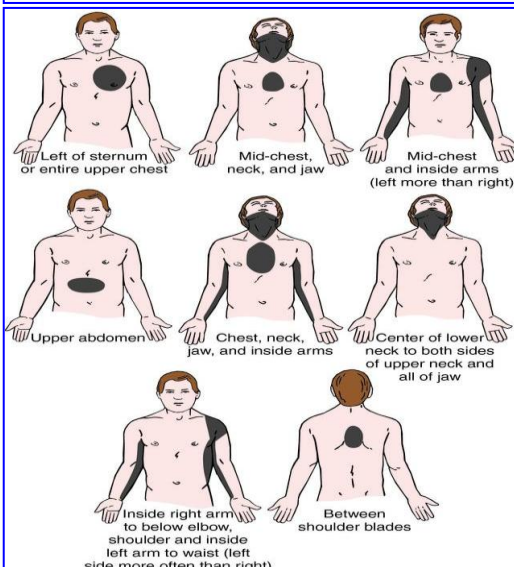
Clinical picture

Symptoms

Pain:

- Onset → acute.
- Course → intermittent.
- Duration → 30 sec. – 30 min.
- Effect of treatment → respond.

Don't forget	
• Analysis : as any pain (11 points with stress on the 3 variants)	
8 as usual	✓ Onset - Course - Duration. ✓ Association. ✓ What ↑ and what ↓. ✓ Effect of treatment. ✓ Date of last attack.
+3	✓ Site. ✓ Radiation. ✓ Character



Site	Retrosternal or precordial
Radiation	Left shoulder, medial aspect of left arm, forearm, hand, neck, lower jaw, epigastrium, right shoulder and back.
Character	Constricting, compressing or in the form of discomfort, tightness oppression or sense of suffocation. (never to be stiching)
Precipitating	Muscular exercise, sexual intercourse Emotional stress الهَم , h heavy smoking, h heavy meals, cold atmosphere (h ypothermia) or h igh altitudes..
Relieving	Rest, sublingual nitrates.
Duration	Few minutes, usually less than 20 min, but may be prolonged.
Association (Other symptoms)	Dyspnea, palpitation, sense of fear of death (angor animi), sweating, dizziness, fainting, nausea, vomiting and eructation at the end of the attack

Angina equivalent: these manifestations without pain and this usually occurs in the elderly.

Signs:1- During the attack

- Bilateral basal rales or arrhythmia.
- Pallor, sweating, tachycardia and hypertension (sympathatic stimulation).
- Muffled 1st heart sound.
- Reversed splitting of second heart sound.
- 4th heart sound due to diminished compliance of the LV.
- MR murmur: due to papillary muscle dysfunction.

2- In between the attacks:

- No signs can be detected.
- Signs of the underlying cause e.g AS, exanthema.

Types of angina

Stable	The Pain is stable as regard duration, severity, character and precipitation in the last 2 months.
Unstable	Includes: • Angina at rest. • Angina of recent onset . • Increase in pain duration, severity, frequency or precipitation at lower threshold (Crescendo angina) • Post infarction angina.
Variant	<u>(Prinzmetal angina, angina inversa, vasospastic angina)</u> • Angina at rest & not precipitated by decreased oxygen demand. • It is usually associated with (S – T segment elevation). • It is most probably due to coronary spasm. (vasospastic angina) • Coronary arteriography may be normal.
Other types	<u>Angina decubitus</u> : Angina that is precipitated by lying down & improved by sitting. <u>Nocturnal angina</u> : Angina that awakens the patient at night. <u>Angina of Lewis</u> : Occurring in severe aortic incompetence e.g. syphilitic characterized by being nocturnal, prolonged & associated with excessive sweating.
Cardiac Syndrome X (microvascular angina)	• It is due to small vessel disease as microangiopathy of DM or in vasculitis and in collagen diseases • The pain occurs with excursion and not at rest • Radioisotopic scanning shows ischemia while coronary angiography is normal.

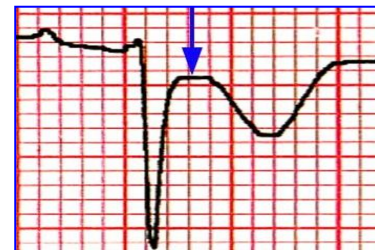
Any other forms of angina classified as unstable angina.

Investigations

A. During anginal pains:

1. ECG:

- ST depression and flat or inverted T-waves.
- In Prinzmetal angina there is ST elevation with hyperacute T wave.
- Arrhythmia.



2. Echocardiography: may show regional wall abnormalities in ischemic areas or MR.

3. To exclude myocardial infarction:

- Normal cardiac enzymes.
- ESR, leucocytic count and C-reactive protein are normal (not done).

B. In between the attacks:

1. ECG:

- Resting ECG: may be completely normal or may show the evidence of the underlying cause.
- Exercise ECG:
 - Done by exercising the patient on Treadmill.
 - Positive test includes:
 - ECG changes of ischemia or arrhythmia.
 - Chest pain during exercise.
 - Hypotension with exercise.
 - Test is limited in patients with:
 - Base line ECG changes e.g. LV strain or LBBB.
 - Patient who cannot exercise e.g. LL ischemia, HF, severe hypertension, unstable angina.
- Holter monitoring (Ambulatory ECG): used for diagnosis of ischemia and correlating chest pain to ECG changes.

2. Radio-isotopic scanning:

- Thallium imaging:
 - Thallium injection during the peak of exercise will distribute the isotope to normal but not ischemic myocardium leading to cold spot in the latter when the heart is viewed by Gamma camera.
 - The cold spot will disappear at rest because of return of blood flow to ischemic zone.
 - If the patient cannot be exercised, the test can be done by dipyridamole or dobutamine administration (pharmacological testing).
- Technetium-99m: Like Thallium it shows cold spot in ischemic areas but reversibility can be shown only by re-injection of another dose at rest

3. Echocardiography:

- May show the underlying cause e.g. AR.
- Exercise or Dobutamine echocardiography may show reversible regional wall motion abnormality.

4. Cardiac catheterization

- **INDICATIONS:**
 - Highly suspicious chest pain with inconclusive non-invasive tests.
 - Unstable angina.
 - Large area of ischemia in perfusion scan.
- **VALUE:**
 - **Diagnostic:**
 - To diagnose The presence of coronary flow limitation.
 - To diagnose the cause of the coronary narrowing e.g. atherosclerosis, osteal stenosis, spasm.
 - To reveal the site and the extent of coronary lesion.
 - To determine the line of treatment in the patient whether medical, coronary intervention or surgery.
 - **Prognostic:**

Prognosis is affected by the number, and the extent of coronary vessels in addition to LV function:

 - With normal LV function the 5 year mortality rate with one, two, and three coronary vessel disease is 2%, 8% & 11 % respectively.
 - Left main artery disease is associated with 15% mortality rate.
 - Mortality is greatly increased if the LV function is impaired.

5. New investigations

- CT angiography.
- PET perfusion scan.
- Magnetic resonance coronary angiography.

6. Investigations for risk factors:

- **Lipid profile:** risk for coronary artery disease is increased if;
 - Elevated cholesterol.
 - Low HDL.
 - Triglyceride level is not directly related to atherosclerosis and their effect may be related to hypercholesterolemia.
- **Blood glucose, uric acid, CBC and coagulation profile.**

Differential diagnosis:

- Differentiation from other causes of chest pain especially
 - ✓ Cardiac causes :- e.g. myocardial infarction & Mitral valve prolapsed
 - ✓ Gastrointestinal e.g. oesophageal spasm, gastro-oesophageal reflux
 - ✓ Musculoskeletal disorders, e.g. cervical spine disease.
- Determination of the type of angina: stable, unstable or variant.

Treatment

A. During the attack:

- Complete bed rest.
- Sublingual nitrates:
 - e.g. Nitroglycerine (Dinitra): 0.5 mg /or Isosorbide dintrate (Isordil) 5 mg.
 - Tablet effect appears after 1 minute, and if no response repeat the dose every 5 minutes till the pain is relieved or a maximum of 3 tablets has been given since the later presents either unstable angina or myocardial infarction and the patient should be transferred to hospital.

B. To prevent the attacks of angina:

- **Avoid precipitating factors** e.g. exposure to cold, heavy meals or emotional stress.
- **Modification of risk factors:**
 - Proper control of blood pressure diabetes mellitus and hyperuricemia.
 - Stop smoking.
 - Correction of dyslipidemia:
 - Diet: avoid heavy meals with excess fat and carbohydrate.
 - Increase intake of vegetables and fruits.
 - Increased intake of omega-fatty acids (antioxidant properties).
 - Reduction of body weight in obese patients.
 - Drugs: These include:
 - 1. Statins:
 - Simvastatin, or atrovastatin (Lipitor)
 - They reduce cholesterol level.
 - Side effects: mainly myopathy & increased liver enzymes.
 - 2. Fibrates:
 - Bezafibrate (Bezalip).
 - They reduce triglyceride level.
 - Side effects: cholestasis, GB stones or myopathy.
 - 3. Nicotinic acid derivatives: They cause flushing and GIT disturbances.
 - 4. Cholestyramine:
 - Reduces the level of cholesterol by chelating bile salts in the GIT.
 - Side effects: GIT disturbances with diarrhea and steatorrhea.
 - 5. Ezetimibe:
 - Reduces intestinal absorption of cholesterol and lowers LDL.
 - Side effects: myopathy and increased liver enzymes.
- **Physical exercise & moderation of life style:**
 - Gradual Increase in physical activity with regular dynamic exercise (walking) short of anginal pain precipitation.
 - Exercise can increase collateral blood flow, reduce platelet aggregation, increase fibrinolytic activity of blood, and reduces BP and heart rate response to exercise.

- Decrease stress by tranquilizers
- avoid excretion by reducing working hours and by adequate night sleep with regular vacations.
- **Anti anginal drugs:**
 - 1. **Nitrates**
 - Mechanism of action:
 - ✚ Activation of Guanylate cyclase enzyme (cGMP) → intracellular calcium → smooth muscle relaxation.
 - Action:
 1. It reduce cardiac oxygen consumption by:
 - ✚ Venodilatation (mainly) with reduction of venous return and preload.
 - ✚ Arteriolar dilatation: with reduction of after load.
 2. Increases oxygen supply by coronary vasodilatation.
 - Preparations:
 1. Nitroglycerine:
 - ✚ Sublingual (Dinitra): 0.5 mg.
 - ✚ Oral (Nitromak) 2.5 mg every 8 hours.
 - ✚ Ointment.
 - ✚ Patches(Nitroderm): 5 or 10 mg patches.
 2. Dinitrate Isosorbide
 - ✚ Sublingual (Isordil): 5 mg tablets.
 - ✚ Oral (Coronit): 10-40 mg tds.
 3. Isosorbide mononitrate:
 - ✚ Oral (Effox) 20-40 mg BID and slow release forms (50 mg once daily).
 - Side effects:
 1. Orthostatic hypotension.
 2. Dizziness and even syncope.
 3. Headache & flushing.
 4. May precipitate angina on withdrawal.
 5. Tolerance(nitrate free interval of 8 hrs may prevent tolerance).

2. Beta blockers

- Mechanism:
 - ✚ Reduction of HR, BP and cardiac contractility with reduction of myocardial oxygen demands.
 - ✚ Reduction of HR and BP response to exercise with improvement in working capacity of the patient.
- Side effects:
 1. Cardiac:
 - ❖ May precipitate heart failure in patients with reduced contractility.
 - ❖ Heart block or other bradycardia may occur.
 - ❖ May increase coronary spasm in Prinzmetal angina.
 - ❖ Sudden withdrawal may cause unstable angina or even infarction.
 - ❖ Hypotension.
 2. Respiratory:
 - ❖ May produce bronchospasm.
 - ❖ Aggravation of bronchial asthma and COPD.
 3. Others
 - ❖ Impotence.
 - ❖ Depression and night mares.
 - ❖ Mask the manifestations of hypoglycemia.
 - ❖ Intermittent claudication.
- Indications:
 - Angina.
 - Arrhythmia.
 - Hypertension.
 - Fallot tetralogy and IHSS.
 - Thyrotoxicosis.
 - Pheochromocytoma: only with alpha blockers.
 - Portal hypertension.
 - Migraine.
- Preparations:
 1. Non-selective β -blockers:
 - ✚ Propranolol (Inderal): 40-240 mg/day.
 - ✚ Nadolol (Corgard): 80-240 mg/day.
 2. Selective β -blockers:
 - ✚ Atenolol (Tenormin): 50-100 mg/day.
 - ✚ Metoprolol (Betaloc): 50-150 mg/day.
 3. Short acting β -blockers:
 - ✚ Esmolol (Breviblock): used as IV form only.

3. Calcium channel blockers

- Mechanism of action:
 - + Reduction of cardiac load by arterial dilatation (afterload) and by reduction of cardiac contractility and heart rate.
 - + Increases coronary blood flow by inducing coronary dilatation.
 - + Anti-Arrhythmic.
- Side effects:
 1. Cardiovascular:
 - ❖ Hypotension.
 - ❖ Bradycardia and heart block with verapamil and diltiazem.
 - ❖ Reflex tachycardia may occur with Nifedipine.
 - ❖ May precipitate heart failure (Only diltiazem and verapamil).
 2. Others:
 - ❖ Headache and facial flushing.
 - ❖ Constipation.
- Preparations
 1. Verapamil (Isoptin): 80-120 mg TDS.
 2. Diltiazem (Altiazem or Teldium): 30-90 mg TDS.
 3. Nifedipine (Epilat): 10-20 mg TDS.
 4. Amlodipine (Norvasc): 5-10 mg once daily.
 5. Nimodipine. (used in subarachnoid hemorrhage).
- Indications:
 - Angina and myocardial infarction.
 - Arrhythmia.
 - Hypertension.
 - Fallot tetralogy & IHSS.
 - Oesophageal spasm.
 - Subarchnoid hemorrhage.

3. Anti-platelets

To decrease incidence of myocardial infarction and sudden death

- Acetyl salicylic acid (Aspirin)
 - 150 - 350 mg once daily.
 - It works by inhibition of PGs synthesis leading to reduced platelet aggregation.
 - Side effects: peptic ulcer and bleeding tendency.
- Ticlopidine (Ticlid)
 - Inhibits platelet aggregation.
 - Dose: 250 mg/12 hrs.
 - Side effects: agranulocytosis and bleeding tendency.

- Clopidogrel (Plavix)
- Tirofiban (Aggrastat): used in unstable angina only.
- Dipyridamole: not used in angina as it causes coronary steal Phenomenon.

Coronary revascularization

1. Percutaneous transluminal coronary angioplasty (PTCA) :

- Procedure:
 - ❖ Balloon inflation in the vessel at the site of stenosis.
 - ❖ Can be done for both native vessels and grafts following CABG.
- Indications:
 - ❖ Unstable angina.
 - ❖ The number of vessels may be 1,2, or even 3 vessels.
 - ❖ The lesion should be non calcified, proximal, short in length and not involving the ostium of the vessel.
- Disadvantages and complications:
 - ❖ Re-stenosis rate after one year is 30%.
 - ❖ Coronary thrombosis in 1 % of cases.
 - ❖ Coronary dissection.
 - ❖ Air embolism.
 - ❖ Retro-peritoneal bleeding.

2. Coronary stenting:

- Indications:
 - ❖ In patients with recurrent coronary stenosis following PTCA.
- Advantages
 - ❖ Higher patency rate than PTCA.
 - ❖ It increases survival rate and event free interval.
- Types:
 - ❖ Regular stents.
 - ❖ Drug eluted stents to reduce the incidence of restenosis.

3. Coronary artery bypass grafting (CABG):

- Procedure:
 - ❖ Coronary lesions are bypassed by a vascular graft from the aorta to the coronary distal to the stenosis
 - ❖ Grafts used are either venous graft (higher rate of stenosis), radial, internal mammary arteries (lower rate of stenosis) or synthetic grafts.

- Indications:
 1. Left main coronary artery disease.
 2. Three vessel disease or more with impaired LV function.
 3. Unstable angina with lesions not suitable for PCI.
 4. Complicated PCI .
- Complications:
 1. Mortality less than 1%.
 2. Occlusion of grafts: 10-20 % in the first year and then 4% / year for the next 4 years.
The reocclusion rate is higher for venous grafts and avoided by using anti-platelets and modifying risk factors.
 3. Post operative myocardial infarction in 5-10% of cases.
 4. Post operative stroke, tamponade, hemorrhage.

4. Others:

- Laser angioplasty.
- Rotablaters.
- Transmyocardial revascularization.

N.B.

- Percutaneous coronary intervention (PCI), commonly known as coronary angioplasty or simply angioplasty.

Acute coronary syndromes

Acute coronary syndromes (ACS) include:

- ST-elevation myocardial infarction (STEMI)
- Non-ST-elevation myocardial infarction (NSTEMI)
- Unstable angina (UA).

The difference between UA and NSTEMI is that in the latter there is occluding thrombus, which leads to myocardial necrosis and a rise in serum troponins or CK-MB. Myocardial infarction (MI) occurs when cardiac myocytes die due to myocardial ischaemia, and can be diagnosed on the basis of appropriate clinical history, 12-lead ECG and elevated biochemical markers – troponin I and T, CK-MB.

Myocardial infarction

Definition

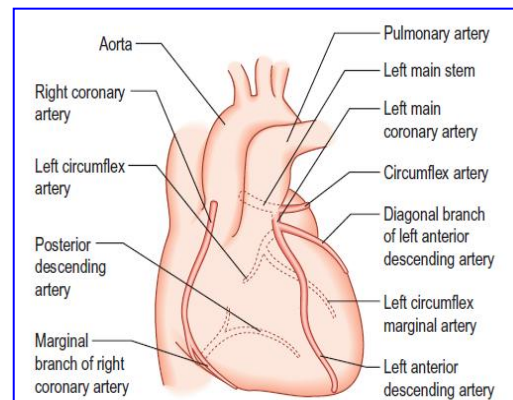
Complete **coronary obstruction** leads to an **ischaemic necrosis** of a part of the cardiac muscle.

Aetiology

Causes of coronary ischaemic heart disease (see above) but usually it occurs as a result of **thrombosis on top of coronary atherosclerosis**.

The site of infarction depends on the occluded artery:

- Occlusion of anterior descending artery: causes anterior infarction.
- Occlusion of circumflex artery: causes lateral infarction.
- Occlusion of right coronary artery: causes inferior infarction.



Clinical picture

(Symptoms + Signs + complications)

- Patients with myocardial infarction may present with chest pain (described as angina pain with differences), complications or both.
- The condition, however, may be asymptomatic.

A. Symptoms

1. Chest Pain

- It is the main presenting symptom.
- It differs from anginal pain in the following features:
 - ✓ It is more severe and usually crushing or squeezing in nature.
 - ✓ It is more prolonged.
 - ✓ It may occur without precipitating factors or after severe mental stress.
 - ✓ It is not relieved by rest or sublingual nitrates.
 - ✓ It may be associated with many complications.

The pain may be absent (painless infarction) Silent myocardial ischaemia

Definition: lack of symptoms in spite of sure ECG or nuclear evidences of ischaemia.

Caused by:

- Autonomic neuropathy especially in diabetics.
- Anesthesia, sedation or impaired consciousness.
- Infarction occurring after cardiac transplantation.
- In old age.
- In transplanted heart.

2. Symptoms of complications e.g. shock, LVF.

3. Symptoms related to the cause e.g. aortic dissection or systemic embolism.

B. Signs**General:**

1. Pulse: usually tachycardia due to pain however the pulse may exhibit:
 - ❖ Arrhythmia.
 - ❖ Bradycardia: as in cases with neurogenic shock or in cases with heart block.
2. Temperature: Mild pyrexia (37.5-38.5) usually develops in the second day & lasts 2-6 days.
3. Blood pressure:
 - ❖ Hypertension due to the effect the pain (sympathetic stimulation).
 - ❖ Shock may occur with reduction of BP by 30 mmHg below the base line of the patient's BP.
 - ❖ Decapitated BP: In hypertensive patients the systolic BP may decrease without drop in diastolic pressure (compliance dependent).
4. Respiration: tachypnea may be present due to anxiety or lung congestion.
5. Face: The patient is usually in severe agony, with pallor and excessive sweating.
6. Other signs related to complications: e.g. congested neck veins in cases of cardiac tamponade.
7. Signs related to cause: xanthelasma, embolism.

Local:

Cardiac examination may not reveal any abnormality or may show:

1. Signs related to complications of myocardial infarction:
 - ❖ Paradoxical pulsations due to LV aneurysm.
 - ❖ Paradoxical splitting of S2.
 - ❖ S3 or S4 gallop due to LV dysfunction.
 - ❖ MR due to papillary muscle dysfunction.
2. Signs related to the cause: e.g. AR in dissecting aortic aneurysm.

C. Complications(5, 5)

Early (5)	Late (5)
1- Shock:- Cardiac - neurogenic	1- Post inf. S.
2- H.F:- LVF - RVF	2- Aneurysm
3- Arrhythmia:- Common - dangerous	3- Thromboembolism
4- Heart: Endo /Myo /Peri-cardium	4- Frozen shoulder
5- Sudden death	5- Post inf. angina

The patient may present by one or more of the following complications:

Early complications:

1. Shock:

- ❖ Cardiogenic shock:
 - Due to: infarction of more than 40% of LV
 - Clinically: there is hypotension with cold, clammy sweat, disturbed sensorium and oliguria (urine < 20 ml/Hr)
 - Investigation: extensive infarction shown by ECG, echocardiography, and radio- isotopic scanning. Cardiac index is below 1.8 L/min/m²
 - Prognosis: high mortality rate (80%) due to reduction of coronary perfusion along with acidosis and arrhythmia that tends to increase myocardial damage.
- ❖ Neurogenic shock:
 - Due to: severe pain, there is intense vagal stimulation leading to bradycardia & hypotension.
 - Prognosis: the condition is easily reversed by analgesics.

2. Heart failure:

- ❖ Left ventricular failure :
 - occurs due to infarction of the LV.
 - The most common.
 - Chronic → low cardiac output and pulmonary congestion .
 - Acute → acute pulmonary edema (no cardiomegaly) .
 - Prognosis → bad.
- ❖ Right ventricular failure :
 - Usually secondary to LV failure or due to RV infarction that complicates infero- posterior infarction in 50 % of cases.
 - Chronic → low cardiac output and systemic congestion.
 - Prognosis → good.

3. Arrhythmia:

- ❖ All types of arrhythmia may occur in myocardial infarction.
- ❖ The most common is → sinus tachycardia and extrasystoles.
- ❖ The most dangerous is → VT and heart block.

4. Heart

- ❖ Endocardium: (Thrombo-embolism)
 - Mural thrombosis: in the LV --- may lead to systemic embolism (e.g. cerebral).
- ❖ Pericardium:
 - Dry pericarditis may occur.
 - Haemorrhagic effusion or haemopericardium may develop.

➤ Post-myocardial infarction pericarditis

occurs in about 20% of patients in the first few days following MI. It occurs more commonly with anterior MI and ST elevation MI with high serum cardiac enzymes, but its incidence is reduced to 5–6% with thrombolysis.

It may be difficult to differentiate this pain from recurrent angina when it occurs early (day 1–2 post-infarct) but a good history of the pain and serial ECG monitoring is helpful. Pericarditis may also occur later on in the recovery phase after infarction. This usually occurs as a feature of Dressler's syndrome (see below).

❖ Myocardium : myocardial rupture may lead to

Direction	Leads to	Manifested by
Outward	Haemo-pericardium	Cardiac tamponade may lead to death.
Inward	Rupture of septum	Acquired VSD
Midway	Rupture of papillary muscle	Acute mitral Regurge --- Acute HF

5. Sudden death:

Due to ventricular fibrillation, ventricular asystole or rupture of the heart.

Late complications :

1. Post-infarction syndrome "Dressler's syndrome":

- ❖ an autoimmune response to cardiac damage occurring 2–10 weeks' post-infarct.
- ❖ Autoimmune reaction to myocardial damage is the main aetiology, and anti myocardial antibodies can often be found.
- ❖ Recurrences are common.
- ❖ Differential diagnosis includes a new myocardial infarction or unstable angina.

2. Myocardial aneurysm:

Presented with	Examined with
• Refractory heart failure. • Recurrent embolism. • Recurrent arrhythmias. • Rupture ---- haemopericardium.	• Cardiac exam.: may show double apex - weak S1 • ECG : persistent elevation of the S-T segment. • Echocardiography or screen:- shows paradoxical P.

3. Thromboembolism:

- ❖ **Mural thrombosis:** in the LV --- may lead to systemic embolism (e.g. cerebral).
- ❖ **DVT:** due to prolonged recumbency & may cause pulmonary embolism.

4. Frozen shoulder:

- ❖ It is stiffness & pain of the left shoulder.
- ❖ Due to reflex arteriolar VC leading to ischaemia & fibrous or due to limitation of movement.

5. Post-infarction angina & reinfarction:

- ❖ Due to affection of other coronary arteries.

Differential diagnosis

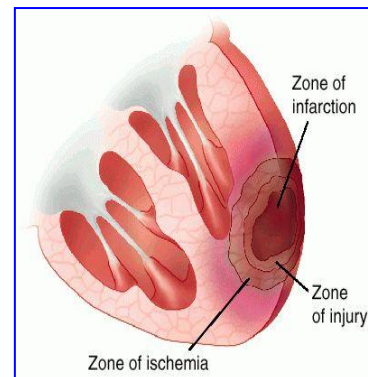
Other causes of acute chest pain (see symptomatology) e.g.

- Unstable angina.
- Massive pulmonary embolism.
- Dissecting aortic aneurysm.
- Dry pericarditis & acute pleurisy.

Investigations

1. ECG:

- ❖ Evidence of infarction:
 - In acute myocardial infarction: ST segment elevation and hyperacute T wave.
 - In old myocardial infarction: Pathological Q wave with flat or inverted T-wave.
- ❖ Site of infarction:
 - Anterior infarction: changes in leads V1 -V4.
 - Inferior infarction: Changes in leads II, III and AVF.
 - Lateral infarction: Changes in leads I, AVL, V5 & V6.
 - RV infarction: Tall R in V1.
- ❖ Complications:
 - Persistent ST elevation for more than 2 weeks in cardiac aneurysms.



2. Cardiac enzymes:

- ❖ CPK (creatine phosphokinase)
 - Source: present in muscles (CK-MM), in brain (CK-BB) and in cardiac muscle (CK-MB).
 - Value:
 - Starts to rise after 6 hrs from myocardial infarction.
 - Peaks after 24 hrs and returns to normal within 2-3 days.
 - (total CK is elevated with > 5% CK-MB isoenzyme).
 - Early peaking occurs with successful reperfusion.
 - May help to estimate the size of the infarction.
- ❖ LDH (Lactic acid dehydrogenase)
 - Source: Present in muscles, RBCs, Kidney, heart and lung.
 - Type :
 - There are 5 isoenzymes (LDH1-5), with LDH1 is specific for the heart.
 - Value:
 - Starts to rise after 12-24 hrs from myocardial infarction.
 - Peaks after 2-3 days and returns to normal within 7-10 days.
 - May help to diagnose patients presenting after 48 hrs when the peak of CK have been missed.

3. Cardiac specific troponin I and cardiac specific troponin T:

- ❖ There level increases in myocardial infarction (after 2 hrs).
- ❖ Remain elevated for 10-14 days.
- ❖ They may help to diagnose infarction in early period and to exclude other causes for CK-elevation.

4. Echocardiography:

- ❖ Infarcted areas appear as akinetic, or dyskinetic segments.
- ❖ Old infarction is usually thinned out.
- ❖ May demonstrate associated complications e.g. cardiac tamponade.

5. Acute infarct scintigraphy:

- ❖ Done to diagnose cases with inconclusive ECG e.g. LBBB, LVH.
- ❖ Technetium-99 [^{99m}Tc] sestamibi is used to detect acute infarcting areas as cold spots.
- ❖ There is no redistribution so imaging can be delayed after injection up to 6 hrs.
- ❖ [^{99m}Tc] pyrophosphate attaches to infarcted areas which appear as hot spot.

6. Non-specific :

- ❖ Complete blood count:
 - Leukocytosis may be within several hours - It peaks in 2 - 4 days - normal after 1 week.
- ❖ Erythrocyte sedimentation rate (ESR):
 - Rises within 3 days - may remain elevated for several weeks.

Treatment of the myocardial infarction

- ✚ Pre hospital (during transfer).
- ✚ Hospital care.
- ✚ After discharge.

1. Pre hospital :

- ❖ Rapid transfer to hospital is a must (Time lost is life lost).
- ❖ Most patients of myocardial infarction die because of arrhythmia particularly VT or heart block. Accordingly, emergency management of myocardial infarction should include using lidocaine (50 -100 mg IV) for tachycardia and atropine (0.5 -1 mg IV) for bradycardia.
- ❖ If the facilities are available, patients indicated for thrombolytic therapy should receive the treatment during ambulance or helicopter transfer.
- ❖ Relief of pain by morphine (2-6 mg).

2. Hospital care :

❖ General measures:

- Hospitalization: Patient should be admitted to CCU where there is continues monitoring and the facilities (pace makers, DC shock) and the experience allows proper intervention to manage complications of myocardial infarction particularly arrhythmia.

- Complete bed rest:
 - There is complete bed rest for 2-3 days.
 - In the absence of complications → the patient is allowed to use bedside commode and can sit twice daily in bed side chair.
 - If course uncomplicated → patient can be discharged from the hospital after 6-11 days.
- Care of bowel: stool softener should be given to avoid straining during defecation.
- Diet:
 - Food should be soft, easily digested and fractionated into multiple small meals in the first 5 days.
 - If there is congestive heart failure salt should be restricted.
 - Coffee, tea and cola are avoided .
 - Smoking completely prohibited.
- Oxygen:
should be given by face mask or nasal cannula at rate 24L/min in order to:
 - Increase O₂ concentration in patients with lung congestion.
 - Increase O₂ delivery in ischemic myocardium.
- Relief of emotional stress: diazepam 2-5 mg TDS
and temazepam may be added 15-30 mg at bed time to induce sleep.
- Relief of pain: morphine 5 mmg IV, or mepridine 50-100 mg IV
- Stress ulcer prophylaxis: using H2-blockers e.g. ranitidine
- Control of associated risk factors: e.g. DM

❖ Re-perfusion measures:

Restoring blood flow in the coronary arteries is the most important goal in management of acute myocardial infarction.

Since most myocardial infarctions are due to sudden thrombotic occlusion, attention has been directed towards dissolving the thrombus either mechanically or pharmacologically.

- Indications:
 - Chest pain less than 12 hrs (maximum benefit can be achieved within the first 6 hrs).
 - ECG: ST segment elevation in two or more adjacent leads or new onset of LBBB.
 - Cardiac enzymes show evidence of recent infarction.
- Value:
Reduction of the infarction size leading to:
 - Reduction of mortality (in hospital and long term).
 - Preservation of ventricular function.
 - Prevention or reduction of cardiac arrhythmia.

➤ Methods:

Thrombolytic therapy

i. **Contraindications:**

- History of cerebrovascular accident.
- Recent (within 6 weeks) invasive or surgical procedure.
- Prolonged (more than 10 minutes) cardiac massage.
- Marked hypertension (more than 180/110 mmHg).
- GIT hemorrhage or peptic ulcer within 3 months.
- Bleeding disorders.

ii. **Drugs:**

▪ Streptokinase:

- ✓ Dose: 1.5 million in 100 ml saline units over one hour
- ✓ Advantage: very cheap
- ✓ Disadvantage:
 - * May cause allergic reactions or anaphylactic shock.
 - * Presence of antibodies may reduce the effectiveness of the drug.
 - * Cannot be reused in the next 6 months because of the high antibody titre.
 - * Bleeding tendency.

▪ Tissue plasminogen activator (TBA)

- ✓ Dose: 15 mg bolus followed by 50 mg over 30 minutes then 35 mg over 1 hour.
- ✓ Advantage: Since it's provided by genetic engineering, there is no allergy or antibody formation. It acts only on fibrin.
- ✓ Disadvantages: Very expensive and also cause high bleeding tendency.

Percutaneous transcoronary angioplasty (PTCA)

- i. **Primary PTCA:** Done if there is contraindication for thrombolytic therapy, and markedly reduces mortality in patients presenting with cardiogenic shock if done within 3 hours from chest pain onset.
- ii. **Rescue PTCA:** done in cases with failed thrombolytic therapy.
- iii. **Facilitated PTCA:** half dose of thrombolytic therapy, followed by PTCA.

Coronary artery bypass surgery

- particularly useful for patients presenting with cardiogenic shock within 4-8 hours from the onset of chest pain.
- This can lower mortality ratio from 70% to 30%.

Other drugs

The following drugs are used following reperfusion measures or as the initial management in patients where reperfusion measures are not indicated or contraindicated.

a. Antiplatelets:

- ✓ Drugs: Aspirin: 150 mg chewed at the time of admission then daily.
- ✓ Value: it reduce mortality by itself and enhances that of thrombolytic therapy.

b. Anticoagulants:

- ✓ Indications:
 - * Following thrombolytic therapy or PTCA to prevent rethrombosis.
 - * As prophylaxis for DVT.
 - * Treatment -of thrombotic complications.
- ✓ Dose: heparin is given
 - * IV Bolus 70U/Kg followed by 15U/Kg/Hr following thrombolytic therapy.
 - * 5000 U SQ every 8 Hrs is used as prophylaxis of DVT.

c. β -Blockers:

- ✓ Value: To reduce incidence of arrhythmia and Relieve pain.
- ✓ Dose: metoprolol 5 mg IV every 5 minute with total 15 mg at the time of admission to be followed by 50-100 mg/day.
- ✓ Contraindications: see angina.

d. Nitrates: (IV nitroglycerin)

- ✓ Value: to relieve pain, and reduces cardiac preload
- ✓ Contraindication: Severe hypotension

e. Angiotensin converting enzyme inhibitors (ACEI):

- ✓ Indication:
 - * Within 24 hrs to all myocardial infarction patients with congestive heart failure.
 - * Patients-with asymptomatic reduction in LV ejection fraction.
 - * Left anterior wall motion abnormality or LBBB.
- ✓ Action: they affect remodeling by reducing LV afterload.

❖ Treatment of complications:**➤ Heart failure:**

The same treatment of heart failure but with the following considerations:

- Digitalis effect is not impressive as it does not affect contractility in ischemic or infarcted myocardium.
- Diuretics may reduce blood volume and CO with reduction in coronary perfusion.
- ACEI and nitrates are useful for both ischemia and HF.

➤ Shock:

- Neurogenic shock: analgesics as morphine.
- Cardiogenic shock:
 - ✓ Oxygen inhalation or mechanical ventilation.
 - ✓ Reperfusion as soon as possible by PTCA or CABG.
 - ✓ Inotropic drugs to raise blood pressure:
 - Dobutamine (2.5 -10 ug/kg/min): inotropic with minimal chronotropic or VC effect.
 - Dopamine (2-10 ug/kg/min): inotropic but with more chronotropic effect
 - Amrinonone and milrinone (5-10 ug/kg/min)
 - Epinephrine
 - Norepinephrine
 - ✓ Intra-aortic balloon counter pulsation.
 - ✓ Left ventricular assist devices.

➤ Pericarditis and Dressler syndrome:

Treatment depends on how to manage pain and reduce inflammation.

Medications commonly used include the following:

- *Aspirin, Ibuprofen and Naproxen.*

If these drugs don't help, the following drugs may be prescribed:

- *Colchicine* is an anti-inflammatory drug that may be used to treat persistent or recurring episodes of Dressler's syndrome. Because of potential side effects, such as diarrhea and abdominal pain, this treatment isn't an option for some people.
- *Corticosteroids* can suppress inflammation related to Dressler's syndrome. They're used only when other treatments don't work, because of the risk of serious side effects and because corticosteroids may interfere with the healing of damaged heart tissues after a heart attack or surgery.

➤ Cardiac rupture and cardiac tamponade: Pericardiocentesis and urgent surgical closure of the tear are the only patient chances for survival.

➤ Cardiac mural thrombi: patient should receive oral anticoagulant for atleast 3 months and echocardiographic follow up for the thrombus.

➤ Arrhythmia: managed according to its type.

➤ Frozen shoulder syndrome: physiotherapy & corticosteroids

3. After discharge

1. Reassurance & rehabilitation "gradual return to normal activity".
2. Treatment of post infarction angina.
3. Treatment of precipitating factors e.g. hyperlipidemia.
4. Treatment of late complications e.g. myocardial aneurysm: aneurysmectomy.
5. Aspirin & Beta blockers "*decrease the risk of post infarction angina & reinfarction*".

Systemic Hypertension

Definition

- It is defined as **persistent** elevation of arterial blood pressure.
- Diastolic BP is higher than **90 mm Hg** and or the systolic BP is higher than 140 mm Hg.
- The measurement of BP should be done at least **twice** on at least two subsequent visits.
- A single measurement of systolic BP ≥ 160 mmHg and/or diastolic BP > 105 is sufficient for diagnosis.
- Under complete mental & physical **rest**.

Classifications of Hypertension

A. Systolic & diastolic hypertension

B. According to aetiology:

1. Essential hypertension.
2. Secondary hypertension.
3. Paroxysmal hypertension.
4. Hypertension in pregnancy.

Isolated systolic hypertension:

- Aortic atherosclerosis.
- Aortic incompetence.
- Hyperdynamic circulation.
- Marked bradycardia.

C. According to clinical presentation

1. Benign, accelerated & malignant hypertension.
2. Uncomplicated & complicated hypertension.
3. Mild, moderate, severe & very severe hypertension.

Causes

The majority (80–90%) of patients with hypertension have primary elevation of blood pressure, i.e. essential hypertension of unknown cause.

Essential hypertension

Essential hypertension has a multifactorial etiology:

- **Genetic factors.**
- **Fetal factors .**
- **Environmental factors :**
 - Obesity.
 - Alcohol intake.
 - Sodium intake.
 - Stress.
- **Humoral mechanism.**
- **Insulin resistance:** Peripheral resistance to insulin has been observed in patients with essential hypertension, this leads to:
 - Na reabsorption at renal tubules
 - Sympathetic activity.
 - Stimulate growth of vascular smooth muscle cells.

Secondary hypertension

Secondary hypertension is where blood pressure elevation is the result of a specific and potentially treatable cause:

➤ **Neurogenic causes**

- ↑ ICP.
- Familial dysautonomia
- Lead encephalopathy.
- Bulbar poliomyelitis.
- Polyneuritis (lead or porphyria).
- Tumors or inflammation in the medulla or hypothalamus.

➤ **Vascular causes**

- Coarctation of aorta.
- Polyarteritis nodosa, Takayasu's syndrome.
- Polycythemia and porphyria.

➤ **Endocrinal causes**

- Pheochromocytoma.
- Conn's syndrome.
- Cushing syndrome.
- Hyperparathyroidism.
- Acromegaly.
- Myxedema.

➤ **Renal causes**

a) Vascular (renal artery stenosis):

- Atherosclerosis.
- Fibromuscular dysplasia.
- Vasculitis.
- Renal artery thrombosis & embolism.

b) Paraneuronal:

- Acute & Chronic glomerulonephritis.
- Chronic pyelonephritis.
- Polycystic kidney.
- Collagen diseases.
- Gout, nephrocalcinosis, amyloidosis.
- Diabetic nephropathy.

➤ **Drugs:**

- Contraceptive pills (estrogen stimulates liver synthesis of angiotensinogen).
- Corticosteroids & ACTH.
- Licorice & carbenoxolone.

➤ **Miscellaneous**

- Toxemia of pregnancy.
- Hypercalcemia.

Clinical Picture

Uncomplicated hypertension

➤ **Symptoms:**

- May be asymptomatic.
- Headache, tinnitus, dizziness.
- Epistaxis.
- Palpitation.
- Symptoms of cause in 2ry hypertension e.g. renal failure.

➤ **Signs:**

○ General:

- **BP:** hypertension has been classified according to severity into:

	Systolic	Diastolic
Mild	140 - 159	90 - 99
Moderate	160 - 179	100 - 109
Severe	≥ 180	≥ 110

- **Pulse:**

- * In systolic hypertension pulse volume is increased.
- * In systolic and diastolic hypertension pulse volume is diminished.
- * Pulsus alternans : indicate LV strain.

- **Funds examination for changes of hypertensive retinopathy**

○ Cardiac examination:

- **Inspection & palpation:**

- * Heaving apex

- **Auscultation**

- * ↑ S2 over aortic area.
- * Aortic ejection click.
- * Systolic ejection murmur due to dilatation of the aortic ring.
- * Apical S4.

○ Signs of cause.

Complicated hypertension

Hypertension may lead to several complications:

➤ **CNS:**

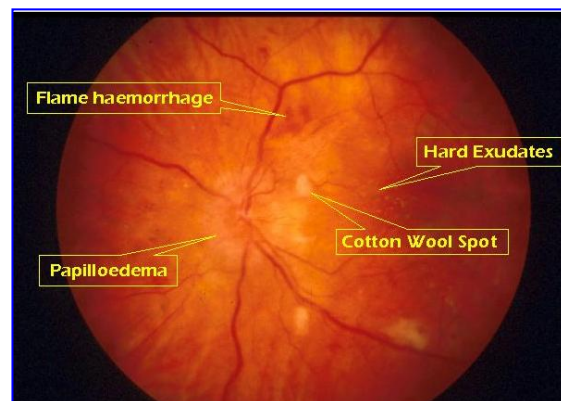
- Transient ischemic attacks.
- Cerebral thrombosis & lacunar infarcts.
- Cerebral hemorrhage due to rupture of vascular microaneurysms .
- Subarchnoid hemorrhage
- Hypertensive encephalopathy:

Mechanism: failure of cerebral auto-regulatory mechanisms with sudden elevation of BP → cerebral oedema.

- CP:**
- Headache, vomiting, blurring of vision.
 - Convulsions & then coma without lateralization.

➤ **Eye:**

- (Hypertensive retinopathy):
Grades (Keith Wagener classification)
 - Grade I: Narrowing of arteries.
 - Grade II: Sclerosis of retinal arteries (Silver wiring) & Kinking of veins at arteriovenous crossing points.
 - Grade III: Flame shaped hemorrhages, fluffy cotton exudates.
 - Grade IV: Papilloedema plus grade III.

➤ **Cardiac complications:**

- Left ventricular hypertrophy and failure.
- Left ventricular diastolic dysfunction.
- Right ventricular failure which is either due to LVF or reversed Bernheim effect (concentric → LVH → bulge of interventricular septum in RV → impairment of RV filling).
- Coronary atherosclerosis leading to cardiac ischemia.
- Arrhythmia: e.g. AF secondary to LV diastolic dysfunction or cardiac ischemia.

➤ **Vascular complications:**

- Accelerated atherosclerosis
- Aortic dilatation & AR.
- Arterial medial hypertrophy and later on fibrinoid degeneration.
- Hemorrhage e.g. epistaxis, haematuria due to rupture of small vessels
- Dissecting aortic aneurysm.

➤ **Renal complications:**

- Proteinuria & microscopic haematuria due to glomerular injury
- Atherosclerosis of the efferent and afferent arterioles and glomerular capillaries → glomerulosclerosis → renal failure in 10-20% of patients.
- Renal artery stenosis.

N.B.

- **Accelerated hypertension**: Diastolic BP > 130 and retinopathy grade III.
- **Malignant hypertension**: Diastolic BP > 130 with retinopathy Grade IV & accelerated organ damage.
- **Labile hypertension**: Fluctuation in BP level between normal and hypertensive readings. These patients are liable for complications of hypertension more than normal individuals and finally they become hypertensive
- **Decapitated BP**: In hypertensive patients the systolic BP may decrease without drop in diastolic pressure (compliance dependent).
- **Hypertensive urgency**: rapid rise of blood pressure > 220 / 120 mmHg and not associated with target organ damage e.g. renal failure, heart failure.
- **Hypertensive emergency**: rapid rise of blood pressure > 220 / 120 mmHg and associated with target organ damage.

Investigation

Investigations for detection of associated risk factors of atherosclerosis :

- Blood glucose level.
- Lipid profile : cholesterol (LDL & HDL) and triglycerides.

Investigations of cause:

These done in cases of secondary hypertension and include:

1. Renal causes:
 - Urea, creatinine
 - Urine analysis
 - Abdominal sonography
2. Cardiac causes: echocardiograph, ECG and catheterization.
3. Endocrinal causes: serum level of adrenaline, cortisol, or Aldosterone.

Investigation for complications e.g.

- Renal function tests.
- Echocardiography, ECG.
- Fundus examination.
- CT brain for patients with neurological manifestations.

Treatment

Aim:

To achieve and maintain systolic BP less than 140 mmHg and diastolic BP below 90 mmHg to reduce the cardiovascular risk of mortality and morbidity.

Method:

- Non pharmacological lines of treatment
- Pharmacological lines of treatment.
- Treatment of the cause.
- Treatment of complications.

Non-Pharmacological therapy

These lines of treatment may be useful in controlling BP in some patients, reducing the dose of antihypertensive medications in others and/or reducing the risk for premature coronary artery disease.

- Weight reduction for obese patients.
- Stop smoking, coffee and alcohol intake.
- Salt restriction in diet, and avoid fatty meals.
- Regular aerobic exercise.
- Avoid stress.
- Relaxation and biofeedback.

Pharmacological therapy**I. Diuretics** (see heart failure page 16)

- Thiazide diuretics.
- Loop diuretics.
- Potassium sparing diuretics.

II. Vasodilators**A- Direct vasodilators:****1- Sodium Nitroprusside (Nipride):**

- Mechanism of action: release of NO with activation of Guanylate cyclase.
- Dose: 0.25 ug/Kg/min by IV infusion.
- Indication: in hypertensive emergencies or afterload reduction in acute pulmonary oedema
- Side effects:
 - Cyanide poisoning when exposed to light.
 - Thiocyanate poisoning in renal failure patients.
 - Hypotension.
 - Nausea and vomiting.
 - Muscle twitches.

2- Hydralazine (Apresoline):

- Mechanism of action: direct smooth muscle dilator.
- Dose :
 - IV 10 - 20mg.
 - Oral 30 mg TDS.
- Indications: Particularly useful in obstetric cases.
- Side effects:
 - VD leads to headache and hypotension.
 - Reflex tachycardia may precipitate angina.
 - Lupus like reaction.

3- Diazoxide (Hyperstat):

- Mechanism of action: direct smooth muscle dilator.
- Dose: 50-150 mg IV bolus to be repeated.
- Indications: Hypertensive emergencies.
- Side effects:
 - VD leads to headache and hypotension.
 - Reflex tachycardia may precipitate angina.
 - Salt and water retention.
 - Hyperglycemia.
 - Hyperuricemia.

4- Minoxidil (Linoten):

- Mechanism of action: direct smooth muscle dilator
- Dose : 25-50 mg/day
- Side effects:
 - VD leads to headache and hypotension.
 - Reflex tachycardia may precipitate angina.
 - Salt and water retention.
 -

B- Angiotensin converting enzyme inhibitors: (see heart failure page 17).

C- Angiotensin receptor blockers: (see heart failure page 17).

D- Calcium Channel blockers: (see angina page 35).

III. Sympathetic blockers

A- Central acting:

- Preparations: Clonidine(Catapress)
- Dose: 0.1 - 0.6 mg daily
- Mechanism of action: Inhibition of hypothalamus and the vasomotor center.
- Side effects:
 - Postural hypotension.
 - Dry mouth, constipation.
 - Rebound hypotension if the drug was stopped suddenly.
 - Insomnia.

B- Ganglion blockers:

- Preparations: Trimethaphan (Arfonad)
- Dose: 1- 6 mg/ min IV.
- Mechanism of action: Inhibition of sympathetic and parasympathetic ganglia.
- Side effects:
 - Postural hypotension.
 - Dry mouth, constipation.
 - Retention of urine.
 - Blurred vision.
 - Impotence.

C- Inhibitors of catecholamine synthesis

- Preparations: Alpha methyl dopa (Aldomet)
- Dose: 250-500 mg TDS
- Mechanism of action: It forms a-methyl norepinephrine that stimulates also the α -2 receptors in the brain with decrease in sympathetic flow.
- Side effects:
 - Hemolytic anemia - Chronic hepatitis - Lupus like syndrome.
 - Ulcerative colitis - Impaired ejaculation - Galactorrhea.

D- Inhibitors of catecholamine release:

- Preparations: Guanethidine (Ismeline)
- Dose: 20 -120 mg/day.
- Mechanism of action: Inhibition of catecholamine release from nerve endings.
- Side effects:
 - Postural hypotension.
 - Dry mouth, diarrhea.
 - Bradycardia.
 - Fluid retention.
 - Impaired ejaculation.

E- Depletion of catecholamine stores:

- Preparations: Reserpine (Serpasil)
- Dose: 0.1-0.25 mg TDS
- Mechanism of action: Inhibition of catecholamine re-uptake in nerve endings.
- Side effects:
 - Depression and nightmares.
 - Extrapyrimalid manifestatons (Parkinsonism).
 - Rebound hypotension if the drug was stopped suddenly.
 - Impotence (reversible).
 - Diarrhea.
 - Water retention.

F- Adrenergic receptor blockers**1- Alpha blockers:**

- Preparations: Prazocin(Minipress)
- Dose: 1- 4 mg TDS
- Mechanism of action: Inhibition of post synaptic α_1 -receptors
- Side effects:
 - First dose hypotension.
 - Dizziness, sedation.
 - Tolerance.
 - Reflex tachycardia.
- Others:
 - Phentolamine and Phenoxybenzamine.
 - Trimazocin.
 - Uradipil.
 - Indoramin (Blocks serotoninergic and H1 receptors also).

2- Beta blockers

- Preparations:
 - ✚ Non selective:
 - * Propranolol (inderal): 40-120 mg/day.
 - * Nadolol (Corgard): 80-240 mg/day.



Selective B1-Blockers:

- * Atenolol (Tenormin): 50-200 mg/day
- * Metoprolol (Betaloc): 50-150 mg/day
- * Esmolol (Breviblock): given IV only

- Mechanism of action:

- Inhibition of SAN, AVN and cardiac contractility.
- Decrease blood pressure.

- Side effects: (see angina page 34).

- Indications: (see angina page 34).

3- Alpha and beta receptor blockers:

- Preparations: Labetalol.
- Dose: 100 - 400 mg/day.
- Side effects: as beta blockers.

4- Others:

- Ketanserin: Selective serotonin receptors blocker
- Rilmendine: Imadoline receptors stimulant
- Tranquilizers: e.g. diazepam 5mg/day.

Choice of the drugs

- ❖ The general role for treatment of hypertension is to start by one drug using the lowest dose needed to control BP.
- ❖ The combination of hypertensive drugs should be used for patients not controlled by monotherapy.
- ❖ There are two methods to follow in choosing the proper antihypertensive drugs:

1. Tailored method:

The choice of anti-hypertensive drugs is individualized according to co-morbidity and associated diseases e.g.:

1. Coronary heart disease and hypertension:

- Beta blockers and calcium channel blockers are used.
- Avoid drugs that produce reflex tachycardia as hydralazine.

2. Heart failure and hypertension:

- ACEI and diuretics are used.
- Avoid drugs that reduce cardiac contractility as beta blockers and verapamil.

3. Diabetes mellitus and hypertension:

- ACEI is particularly useful in reducing the microalbuminuria in these patients.
- Avoid diuretics and diazoxide that may induce hyperglycemia.
- Avoid beta blockers that may mask hypoglycemia in these patients.

4. Hypertension with renal impairment:

- Alpha-methyl dopa is used as it increases renal blood flow.
- Avoid ACEI and K-retaining diuretics.

5. Hypertension with pregnancy:

- In mild cases: salt restriction.
- In pre-eclampsia: bed rest, salt restriction and hydralazine or methyl dopa.
- In eclampsia: as pre-eclampsia but with IV hydralazine and pregnancy termination if there is no response to treatment.

The most commonly used drugs in this method seem to be the following:

Drug	Indication	(with caution) Contraindication
ACEI	▪ Heart failure. ▪ Diabetes mellitus.	▪ Renal artery stenosis. ▪ Renal failure. ▪ Pregnancy.
Beta blockers	▪ Coronary heart disease. ▪ Arrhythmia.	▪ Heart failure. ▪ Bronchial asthma. ▪ COPD. ▪ Diabetes mellitus.
Calcium channel blocker	▪ Coronary heart disease. ▪ Diabetes mellitus.	▪ Heart failure.
Diuretics	▪ Heart failure.	▪ Diabetes mellitus. ▪ Gout.

II. Stepped care method:

This method of treatment depends on the level of blood pressure and planned choice of the drugs depending on the degree of hypertension.

1. Step I:

- Diuretic.
- or Beta blockers.
- or ACEI.
- or Ca-channel blocker.

2. Step II:

- Diuretic + Beta blockers.
- Diuretic + ACEI.
- Diuretic + Ca-channel blocker.

3. Step III:

- Diuretic + ACEI+ Beta blockers.
- Diuretic + ACEI+ Ca-channel blocker.

4. Step IV:

- Add hydralazine or a-blocker to step III.

These steps are followed as follows:

- In mild hypertension: start by step I.
- In moderate hypertension: start by step II.
- In severe hypertension: start by step III.

Treatment of the cause

This is used for the treatment of secondary hypertension e.g.

- 1- Surgical removal of Pheochromocytoma.
- 2- Angioplasty for renal artery.
- 3- Thyroxin therapy for Myxedema.

Treatment of complications**I. Treatment of hypertensive emergencies:**

The initial management should depend on lowering BP to a level that maintains adequate cerebral and coronary perfusion(i.e. 90 mmHg diastolic).

- Patient is better to be admitted to ICU for continuous cardiac monitoring.
- Drugs:
 - Sodium Nitroprusside: : 0.25 -10 ug/kg/min IV infusion.
 - Nifedipine: 10 - 20 mg sublingual.
 - Hydralazine: 10-20 mg IV.
 - Aldomet: 250 mg IV.
 - Diazoxide: 50 - 100 mg / 5 - 10 min IV.
- Oral anti hypertensive drugs should be started after stabilization.
- Venosection is rarely needed in resistant cases.
- Specific treatment for each emergency:
 - Hypertensive encephalopathy:
 - Cerebral dehydrating agents: IV mannitol.
 - Magnesium sulphate per-rectum.
 - Anticonvulsant: IV diazepam and phenytoin.
 - Subarchnoid hemorrhage and Cerebral hemorrhage (see Neurology).
 - Acute pulmonary edema (see page 23).
 - Dissecting aortic aneurysm.
 - Acute renal failure (see nephrology).
 - Papilloedema & retinal hemorrhage

II. Treatment of other complications: e.g.

- Renal failure.
- Heart failure.
- Angina.

Renal Artery Stenosis

Definition

is a narrowing of arteries that carry blood to one or both of the kidneys.

Aetiology

- Atherosclerosis.
- Vasculitis e.g. polyarteritis nodosa.
- Renal artery thrombosis & embolism.

Clinical picture

Renal artery stenosis should be suspected in:

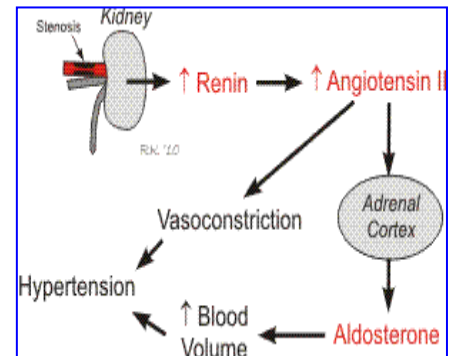
- Young patient less than 25 years.
- Of Sudden onset, severe and resistant HTN
- Bruit in the loin or lateral to the umbilicus.
- Evidence of other occlusive arterial disease.
- Hypertension not responding to medical treatment.

Investigations

- Isotopic scanning.
- Renal arteriography.

Treatment

- Control of hypertension.
- Surgical revascularization.
- Balloon dilatation, may be done for some patients.



Pulmonary hypertension

Definition

Elevation of pulmonary arterial systolic pressure above 30 mm Hg and mean pressures above 20 mm Hg.

Causes

A. **Primary pulmonary hypertension (PPH):**

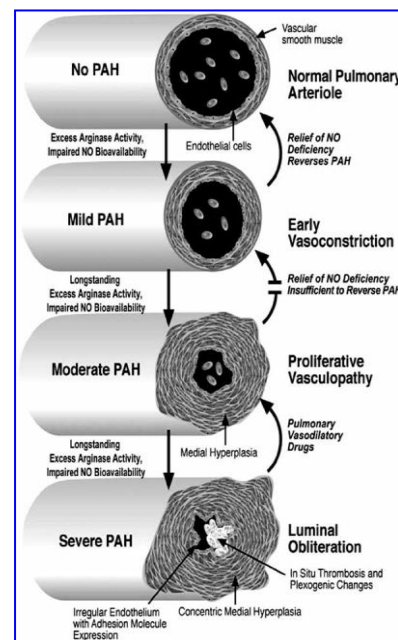
It occurs more commonly in middle-aged females with repeated pregnancies.

Theories:

1. **Endothelial dysfunction:** (Prostacyclin and nitrous oxide endothelin)
2. **Thrombosis in situ.**

B. **Secondary hypertension**

1. **Passive:** due to P congestion --- LSHF - MS.
2. **Hyperdynamic:** increased pulmonary blood flow ---
In congenital left to right shunt e.g. ASD, VSD & PDA.
3. **Vasoconstrictive:** It occurs in
 - Long standing passive pulmonary hypertension.
 - Long standing hyperdynamic pulmonary HTN
 - Hypoxia: e.g. RF (COPD, IPF, pulmonary A-V fistula and high altitudes)
4. **Obstructive (Obliterative)** of pulmonary arterioles:-
 - Long standing vasoconstrictive.
 - Diseases of the pulmonary arteries :- e.g.
 - Recurrent pulmonary emboli.
 - Pulmonary bilharziasis
 - Pulmonary vasculitis e.g. SLE, scleroderma.
 - Pulmonary thromboses in hypercoagulability e.g. sickle cell anaemia.
5. **Acute pulmonary hypertension:-** It occurs in :-
 - Massive pulmonary embolism.
 - Massive lung collapse.
 - Tension or bilateral pneumothorax.



Clinical picture

Symptoms

- Symptoms of low COP.
- Symptoms of right ventricular failure may occur lately.

Signs

General signs:

- Signs of low CO.
- Giant A wave (Or, **Prominent V wave in acute right ventricular failure (functional T.R.)**)
- Signs of right ventricular failure may occur lately.

Cardiac signs:**1. Precordial signs:**

- **Signs of pulmonary hypertension** (in the 2nd left space).
- Pulsations (palpable pulmonary component of S₂) (diastolic shock) & dullness.

2. Auscultation:

- **Heart sounds:** Accentuated P2 with closed splitting
- **Additional S:** Ejection click and S4 over tricuspid area
- **Murmur:**
 - ✓ Early diastolic murmur (Graham-Steell) due to functional **PR**.
 - ✓ Ejection systolic murmur due to relative **PS**.
 - ✓ Functional **TR**.

Investigation**1. Chest X-ray**

- Dilatation of pulmonary arteries in the hila with attenuation of periphery.
- Right atrial & ventricular enlargement.
- Features of the cause may be detected.

2. ECG

- P Pulmonale.
- Right ventricular enlargement.

3. Echocardiography & Doppler ultrasound:-

- Detection of right ventricular enlargement.
- Estimation of pulmonary arterial pressure.
- Features of the cause may be detected e.g. MS.

4. Cardiac catheterization:-

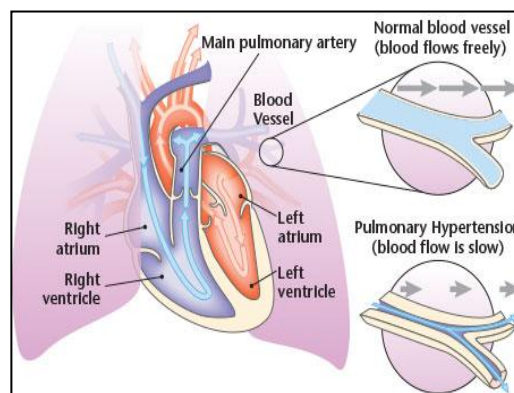
- As, Echo+ Measurement of pulmonary wedge pressure.

Reversibility of pulmonary hypertension

- Response to Vasodilator through Doppler or Catheterization, or
- Ratio: pulmonary / systemic BP.

Treatment

- Treatment of the underlying causes.
- Direct vasodilators, Ca blockers & ACE inhibitors is usually of limited effect.
- Treatment of right sided heart failure.
- Anticoagulants for prophylaxis against secondary thromboembolism.
- Heart lung transplantation is the only radical treatment.



Rheumatic fever

Definition

an inflammatory disease that occurs in children and young adults (the first attack usually occurs at between 5 and 15 years of age) as a result of infection with group A streptococci.

Mechanisms

1. **Antigenic similarity: (Cross-reactivity)**

Antibodies formed against streptococcal antigens react with human tissue antigens since both are immunologically similar.

2. **Altered antigenicity (haptens like)**

Binding of streptococcal antigens to human tissue antigens, renders them antigenic and stimulates antibody formation against them

The latent period usually 1-4 weeks but in chorea it may be longer (several months).

Predisposing factors

- **Age:** 5-15 years (rare below 4 years & above 26 years).
- **Sex:** equal in both sexes except rheumatic chorea which is more common in females.
- **Familial tendency:** due to both hereditary predisposition & similar environment.
- **Socio-economic factors:** more common in developing countries & low social classes.
- **Geographic distribution:** more common temperate zones
- **Seasonal incidence:** more common in winter.

The inflammatory process

A. **Site:** (*Rheumatic fever bites the heart and licks the joints*).

1. Joints: synovial membrane.
2. Heart: endocardium, myocardium & pericardium.
3. CNS: especially basal ganglia.
4. Other serous membranes (pleura) + Skin & subcutaneous tissue, Small blood vessels.

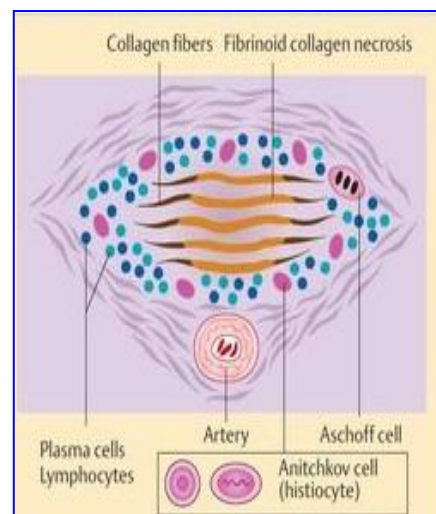
B. **Types of inflammatory reactions:**

➤ **Exudative lesion :**

- Affecting mainly the serous membrane.
- Heals mostly without scarring or deformity.

➤ **Proliferative lesion :** formation of Aschoff nodules

- Affecting mainly the heart and the skin.
- Heals by fibrosis.
- Consists of : (from in to out)
 - Fibrinoid degeneration.
 - Lymphocytes and plasma cells.
 - Aschoff giant cells.
 - Fibroblasts.
 - Layers of fibrosis.



Clinical pictures

A. **Major criteria** "Mnemonic: C.A.N.C.E.R"

- Carditis.
- Arthritis.
- Subcutaneous Nodule.
- Chorea.
- Erythema marginatum.

B. **Minor criteria** "Mnemonic: P.E.A.C.E"

- Prolonged PR interval in the ECG: (**> 0.22 sec due to myocarditis**).
- ESR: elevated.
- Arthralgia: painful joints without swelling.
- CRP (C reactive protein): elevated.
- Elevated temperature "fever".

A. **Major criteria**

1. **Poly arthritis:**

- Number of joint affected :- **polyarticular** (more than 5)
- Distribution: big joints (e.g. knees, ankles, wrists) in a **migratory** character.
- Description: **Arthritis** :- painful, tender, red, hot, swollen, with limitation.
- Drugs: usually subsides spontaneously or dramatically response to **salicylates**.
- Deformity: **no any residual lesion** The joint left (completely free after few days).

jaccoud's arthropathy is a rare late complication.

2. **Rheumatic Sydenham's Chorea:** (See Neurology)

3. **Carditis (Pancarditis)**

Myocarditis	Endocarditis	Pericarditis
Precordial examination: • Cardiac enlargement • Heart failure:- in severe cases • Different types of arrhythmias & HB may occur. • Disproportionate tachycardia Auscultation • Weak heart Sounds. • Ventricular gallop. • Murmurs of functional mitral & tricuspid incompetence. • Tic- Tac rhythm.	Rheumatic valvulitis:- more in left side (M then A) Carey – Coombs murmur:- relative MS in the acute stage only - explained as edematous cusps. disappears after the acute stage. Rare:- Acute MR or AR during the acute stage due to damage of the cusps. Later on:- fibrosis of the valves may lead to stenosis, incompetence or both.	i. Dry Pericarditis: common. ii. Effusion: may occur. iii. Adhesive pericarditis: Rare. v. Constrictive pericarditis: never.

Presence of pericarditis denotes severe pancarditis.

N.B. Arthritis is more common in adults - **Carditis** is more common in children.

4. Subcutaneous nodules:

- Size: Small firm nodules (0.5-2 cm in diameter)
- Site: They tend to be symmetrical and occur over bony prominences & tendons.
e.g.: extensor surfaces of elbows, knees & ankles occiput and spinous processes of thoracic & lumbar vertebrae.
- Shape: Small firm nodules not painful, not tender & not adherent to the skin.

N.B. They are usually associated with carditis.

5. Erythema marginatum:

- Size: variable, the margin progress & the center clears
- Site: These areas appear over the trunk & proximal parts of extremities.
- Shape: areas of erythema non-pruritic, non-painful, not indurated & blanch on pressure.



N.B. They are evanescent & recurrent carditis.

Some say **JONES crITERIA**:

Major criteria:	Minor criteria:
➤ <u>J</u>oint (arthritis). ➤ <u>O</u>bvious (Cardiac). ➤ <u>N</u>odule (Rheumatic). ➤ <u>E</u>rythema marginatum. ➤ <u>S</u>ydenham chorea.	➤ <u>I</u>nflammatory cells (leukocytosis). ➤ <u>T</u>emperature (fever). ➤ <u>ESR</u>/CRP elevated. ➤ <u>R</u>aised PR interval. ➤ <u>I</u>tself (previous Hx of Rheumatic fever). ➤ <u>A</u>rthralgia.

B. Minor Criteria:

- Fever.
- Arthralgia.
- History of rheumatic fever or evidence of rheumatic heart disease.
- Acute phase reactants: High ESR - C-reactive protein - PNL leucocytosis.
- Prolonged P-R interval more than 0.22 sec.
- Other manifestations of rheumatic fever: Pallor, sweating, weakness & loss of weight, Epistaxis, Tachycardia, Pleurisy & Pneumonia.

C. Complications of Rheumatic fever

Early	Late
• Heart failure. • Arrhythmias. • Heart block.	• Rheumatic valvular lesions. • Rheumatic activity. • Rarely: adhesive pericarditis & jaccoud's arthropathy.

Investigations

A. Laboratory investigations

1. Blood picture
 - Polymorph nuclear leucocytosis.
 - Normocytic anemia.
2. Erythrocyte sedimentation rate: Markedly elevated (it is not specific for rheumatic fever) but it is one of the minor criteria & also used for follow up.
3. C-Reactive protein: Non-specific

B. Investigations for streptococcal

1. **Anti-Streptolysin O titer (ASOT)** Elevated in 80% of cases.
 - Titers greater than 250 Todd units/ml in adults & 350 Todd units /ml in children are indicative of recent streptococcal infection.
 - A rising titer may be detected early in the course of rheumatic fever.
2. **Streptozyme test**: positive in more than 95% of cases.
3. Throat culture: positive in only few cases.

C. Cardiac investigations: may show

X-ray	• Chamber enlargement. • Pulmonary vascular (left sided failure). - Pleural effusion
ECG	• Prolonged P-R interval more than 0.33 sec. • May be inverted T wave. • Different types of arrhythmias & heart block may occur.
Echo & Doppler	• Chamber enlargement. • Valve incompetence. • Function (ejection fraction).

Diagnosis of rheumatic fever

Revised Jones Criteria

The diagnosis requires the presence of :

- 2 Major criteria, or
- 1 major & 2 minor criteria.

Plus evidence of recent streptococcal infection e.g.

- ✓ Increased anti-streptococcal antibodies e.g. ASOT.
- ✓ Positive throat culture for group A streptococci
- ✓ Rapid streptococcal antigen test.
- ✓ Recent scarlet fever.

Differential diagnosis

1. Causes of fever in a cardiac patient (infective endocarditis).
2. Causes precipitating heart failure.
3. Causes of acute arthritis e.g. juvenile chronic arthropathy, SLE, Acute leukemia, sickle cell anemia, Henoch Schonlein purpura.

Treatment

Prophylactic:

A. Primary prevention:

- Early diagnosis & proper treatment of upper respiratory tract infections.
- Tonsillectomy may be indicated in some patients with recurrent tonsillitis.

B. Secondary prevention:

- Long acting penicillin (Benzathine penicillin G): 1.200.000 units IM Injection monthly until the age of 25 years, for 5 years after the last attack, or for life.
- Erythromycin (for penicillin-allergic patients: 250mg /6hs.

Therapeutic:

1. General

- Complete bed rest until clinical & laboratory findings (e.g. ESR) return to normal.
- Salt restriction if there is heart failure
- Light nutrient diet

2. Symptomatic treatment: e.g.

- Treatment of heart failure.
- Treatment of chorea.

3. Specific

- Antibiotics: For eradication of the streptococcal infection
 - Benzathine penicillin G 1.200.000-600.000 units IM or - Procaine penicillin: 600.000 units/day for 10 days
 - Erythromycin (for penicillin-allergic patients 250 mg/6 hours orally for 10 days.

➤ Anti-inflammatory drugs

	Aspirin	Steroid
Indication:	- Rh. fever without or with mild carditis - Side effects or contraindications for steroids - During & after withdrawal of steroids	- Rheumatic carditis - If salicylates are not effective or not tolerated
Initial dose: Then:	90 - 120 mg / Kg / day <u>After a satisfactory response is obtained</u> (usually after 3 /weeks) The dose is reduced to 60 - 70 mg / kg / day for an additional 6-9 weeks.	2 mg/kg/day <u>After 3 weeks</u> steroids are withdrawn gradually over an additional 3 weeks.

(Aspirin should be given during withdrawal of steroids & after discontinuation for 3 weeks)

4- Treatment of complication: e.g. Treatment of heart failure.

ENDOCARDITIS

Definition

Inflammation of the endothelial lining of the heart.

Types

1. **Infective endocarditis:** inflammation results from an infecting organism.
2. **Non infective endocarditis:** usually results from sterile inflammation as in collagen diseases e.g. verrucous endocarditis in SLE.

INFECTIVE ENDOCARDITIS

Classification

- According to the causative organism:
 - Bacterial endocarditis.
 - Non-bacterial endocarditis.
- According to the clinical presentation:

Subacute bacterial endocarditis (SBE)	Acute bacterial endocarditis (ABE)
- insidious onset	- a severe disease - acute onset & progressive
- affecting diseased hearts	- affecting diseased or normal hearts
- caused by low virulence organism	- caused by highly virulent organisms

- Other classification of Infective Endocarditis:

Native Valves	Community-acquired native-valve endocarditis. The commonest form after Mitral-valve prolapsed
Prosthetic Valves	Prosthetic – valve endocarditis 15 % of cases of infective endocarditis - may indicate re-replacement
Nosocomial	10 % of all cases of endocarditis - hospital acquired
Infective endocarditis in immunocompromised patients	

N.B.

- *The development of infective endocarditis requires the combination of two factors:
Bacteraemia + underlying cardiac lesion*

Bacteraemia

❖ The causative organisms.

- **Gram-positive cocci:**
 - Strept. viridians (the commonest).
 - Strept. Faecalis & Staph. aureus.
- **Gram-negative bacilli: e.g.**
 - Haemophilus, Actinobacillus, Cardiobacterium, Eikenella & Klebsiella.
- **Pseudomonas & Brucella**
- **Other organisms:** Fungi, Rickettsia, Chlamydia
- **HACEK organisms:** (haemophilus species (Haemophilus parainfluenzae), Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella C.).

Route	Possible organism
Dental, oral & URT	Strept. Viridans
GIT & genitourinary	Strept. faecalis
Cardiac & catheter	Staph aureus

Underlying Cardiac Lesion

❖ **Valvular lesions:**

- Especially MR, AR & AS.
- Mitral valve prolapse: 5 - 8 times more than normal valve.
- The disease is uncommon in MS because of marked fibrosis & low pressure.

❖ **Congenital anomalies:**

- Especially VSD, PDA & A. coarctation.
- The disease is rare in ASD due to low pressure gradient between the two atria.

❖ **Prosthetic valves:**

Two factors in cardiac lesions:

1. *High pressure source:*
 - It is more common in the LS of the heart.
 - It is uncommon in mitral stenosis, AF & ASD.
2. *Narrow orifice:* e.g. more common in smaller VSD.

Endocarditis:

A. *Vegetation formation:-*

- Over the valvular or mural endocardium.
- Composed of bacteria with blood cells & fibrin.
- May detach leading to embolization.

B. *Acute infection* may result in Damage & perforation of the cusps or chordate tendinae.

N.B. *The (myocardium) may be inflamed*

Clinical picture

❖ ***General:***

- Low grade fever except in acute cases.
- Anorexia, malaise and fatigue are the usual initial presentations.

❖ ***Extremities:***

- Loss of pulsations due to embolization (embolization of large vessels occurs usually with fungal endocarditis).
- Mycotic aneurysms may be felt as arterial beads.
- Linear splinter hemorrhages under the nails due to capillary rupture secondary to embolization.
- Osler's nodules: intracutaneous, tender, purple nodules, in the pad of fingers or toes. These result from immunological reaction with swelling of the capillary endothelium and perivascular cellular infiltrates.



- Janeway's nodules: subcutaneous nodules in the palm and sole. They are painless, hemorrhagic nodules.
- Clubbing.



❖ **CNS:**

- Septic encephalopathy.
- Localized neurological deficits due to cerebral embolism.
- Brain abscess (from embolization by septic vegetations).
- Meningitis or encephalitis.
- Subarachnoid hemorrhage due to rupture of mycotic aneurysms (aneurysms resulting from embolization of vasa-vasorum).

❖ **Ocular:**

- Sudden blindness (embolism of central retinal artery).
- White areas of retinal infarcts surrounded by areas of hemorrhage detected by fundus examination (Roth's spot).
- Petechiae and splinter hemorrhages in the conjunctiva due to capillaritis.

❖ **Face:**

- Toxic facies.
- Pallor due to anemia.
- Petechiae in palate and buccal mucosa.

❖ **Lung:**

- Septic pulmonary embolism may lead to lung infarction, lung abscess or recurrent pneumonia.
- This is common in infective endocarditis of the Rt. side of the heart (more in addict patients and in those with Rt. to Lt. shunts).

❖ **Heart:**

- Manifestations of the underlying cause.
- Change in the character of already present murmur or the appearance of new murmur (regurge murmurs resulting from valve perforation or rupture of chordae has a characteristic sharp sound called sea gull murmur).
- Heart failure due to valve damage and myocarditis.
- Aortic root abscess may occur and results in:
 - Conduction abnormalities and bradycardia.
 - Rupture into pericardial sac leading to purulent pericardial effusion.
 - Rupture of IVS with development of VSD.
- Cardiac infarction due to coronary embolization.

❖ **Renal:**

- Glomerulonephritis: induced by immunological reaction to infecting organisms. May lead to renal failure.
- Renal infarction: leading to loin pain, tenderness and hematuria.
- Vasculitis resulting in hematuria and flea bitten kidney.

- ❖ **Mesenteric vascular occlusion:** embolic infarction of the intestine with abdominal pain and GIT bleeding.
- ❖ **Splenic:**
 - Splenomegaly in 30% of cases due to toxemia and immunological reaction.
 - Embolic Splenic infarction with stitching Lt hypochondrial pain and splenic rub.
 - Hepatosplenomegaly may occur with coxiella burnetti endocarditis.

Complications

1. Heart failure due to: valvular damage, myocarditis.
2. Embolization: (see above).
3. Complications of therapy.
4. Perivalvular extension of infective endocarditis.
5. Prolonged Fever.

Investigations:

A. Laboratory investigations:

- **Blood culture:**
 - It is the most important investigation & is positive in about 85% of cases.
 - 6 samples of blood are taken.
 - Samples are cultured under aerobic & anaerobic conditions.
 - The results are looked for after 3 days & up to 60 days.

Bone marrow culture may be done if blood cultures are repeatedly negative.

- **Blood picture:**
 - Normocytic anemia.
 - Polymorph nuclear leucocytosis in **ABE**.
 - Leucopenia may occur in **SBE**.
- **ESR & C - Reactive protein:**
 - Increased but not as markedly as in rheumatic fever.
- **Urine analysis:**
 - Microscopic or macroscopic haematuria Proteinuria & casts.
- **Immunologic tests:**
 - Increased gammaglobulins.
 - False positive rheumatoid factor and serological tests for syphilis.

B. Cardiac investigations:

- **Echocardiography: (transoesophageal echo for small vegetation visualization).**
 - Detects the underlying cardiac disease.
 - Detects complication of endocarditis e.g. mitral & aortic incompetence.
- **Chest X-ray & ECG:**
 - May help in diagnosis of the underlying cardiac disease.

Differential Diagnosis

1. Causes of fever in a cardiac patient especially rheumatic fever.
2. Causes of embolization.
3. Fever of unknown origin.

Duke's criteria for the diagnosis of infective endocarditis :

Major criteria	1) Positive blood culture for infective endocarditis 2) Echo evidences of IE: vegetations - Abscess - New valve regurgitation.
Minor criteria	1) Predisposing factor: known cardiac lesion 2) Fever > 38 3) Echocardiogram : may be some findings but not as major. 4) Microbiological evidence: positive blood culture not as major criterion 5) Immunological : Roth spots , Osler nodes , GN , Rheumatoid factor. 6) Serological studies support an infection. 7) Vascular: emboli , mycotic aneurysm, conjunctival hemorrhage, Janeway L.
<u>Clinical criteria for infective endocarditis requires:</u> ▪ Two major criteria, or ▪ One major and three minor criteria, or ▪ Five minor criteria	

Treatment

Prophylactic

- A. Correction of predisposing heart diseases: whenever possible
- B. Prophylactic antibiotics:

Procedure done	Drugs	Before	After
Dental, oral, or U.R. surgery	Amoxicillin	3 gm 1 hour	1.5 gm 6 hours
	Erythromycin (Penicillin allergy)	1 gm 1 hour	0.5 gm 6 hours
GIT, or genitourinary	Ampicillin 2 gm Gentamycin 80mg IM-IV	1 hour	6 hours
Heart s or catheter	Cefotaxime	2 gm, IV 2 hours	1gm / 6hours for 4doses

Therapeutic

1. Medical treatment:

- Once infective endocarditis is suspected, treatment should be started immediately.
- Treatment should be continued for at least 6 weeks or longer in severe cases.

a. Antibiotic regimens include:

- **For S.B.E.:** (usually due to *Strept. Viridians & faecalis*):
Soluble Penicillin G 2-4 million units / 6h IV or Ampicillin 2 gm / 4h IV
Plus gentamycin: 80 gm/8 h IM.
- **For ABE:** (usually due to *Staph. aureus & Gram – negative bacilli*)
As in SBE with addition of Cloxacillin: 2 gm/4 h IV.

N.B.

For penicillin-allergic patients: Vancomycin is given.

- **Resistant cases:** Should be treated according to results of blood culture.
- **If fungal endocarditis is suspected:** (Amphotericin B given)

b. Symptomatic treatment:

- Rest: complete rest in bed until clinical manifestations improves.
- Diet: light nutrient diet
- Fever: e.g. antipyretics
- Treatment of complications: e.g. Heart failure Renal failure

N.B.

- *Anticoagulant therapy: has not been shown to prevent embolization in infective endocarditis and may increase the risk of intra-cerebral hemorrhage (Q; mycotic aneurysm).*

2. Surgical treatment:**Indications:**

- Valve replacement in : severe valvular damage.
- Replacement of infected prosthesis.
- Excision of infected ductus arteriosus or coarctation.

N.B.

- Any underlying infection should be treated e.g. **dental abscess.**

Differentiation between Rheumatic Fever & Infective Endocarditis:

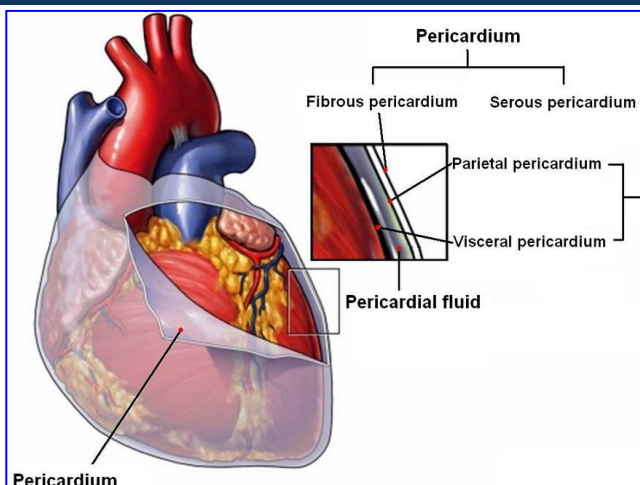
	<i>Rheumatic fever</i>	<i>Infective endocarditis</i>
<i>Fleeting arthritis</i>	+Ve	-
<i>Pericarditis</i>	+Ve	-
<i>Chorea</i>	+Ve	-
<i>S.C. nodules</i>	+Ve	-
<i>Erythema marginatum</i>	+Ve	-
<i>Petechiae</i>	-	+Ve
<i>Clubbing</i>	-	+Ve
<i>Osier's nodules</i>	-	+Ve
<i>Splinter haemorrhage</i>	-	+Ve
<i>Absent pulse</i>	-	+Ve
<i>Splenomegaly</i>	-	+Ve
<i>Kidney manifestations</i>	-	+Ve
<i>Embolic manifestations</i>	-	+Ve
<i>Blood culture</i>	-	+Ve
<i>echocardiography</i>	No vegetations	Vegetations

PERICARDIAL DISEASES

The pericardium acts as a protective covering for the heart. The word "*pericardium*" means around the heart.

It consists of an outer fibrous pericardial sac and an inner serous pericardium made up of the inner visceral epicardium that lines the heart and great vessels and its reflection the outer parietal pericardium that lines the fibrous sac.

The normal amount of pericardial fluid is 20 – 49 mL that lubricates the surface of the heart.



Dry pericarditis.	FAHM + Pain + Rub
Pericardial effusion.	
Constrictive pericarditis	HD changes: S. congestion + Pressure manifestation + P
Adhesive pericarditis.	No HD changes

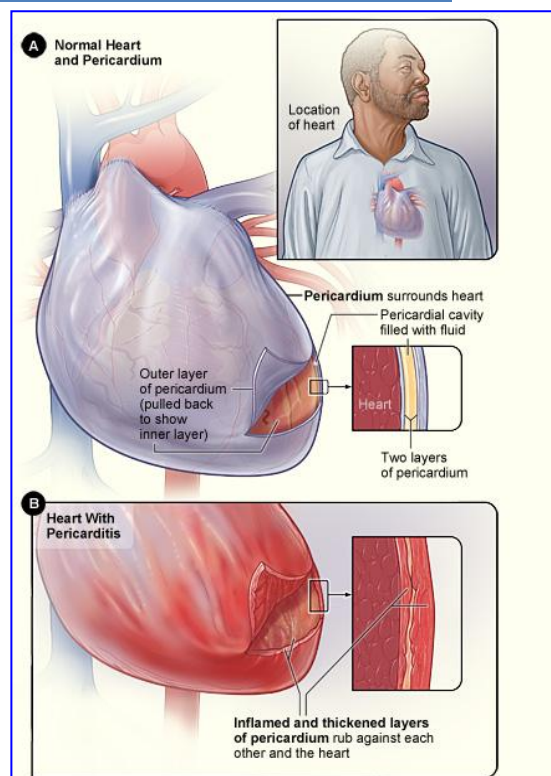
Dry pericarditis

Definition

Inflammation of the pericardial sac. (*the pericardium*)

Etiology

- 1- Idiopathic.
- 2- Post-pericardiotomy.
- 3- Myocardial infarction:
 - Within 2-3 days of infarction due to irritation by necrotic myocardium.
 - After few weeks due to Dressler syndrome.
- 4- Collagen diseases e.g. Rheumatic fever, SLE, Rheumatoid arthritis.
- 5- Traumatic.
- 6- Infective:
 - Viral: as Coxsackie B or Echo virus, which causes pericarditis in any age, and may be recurrent. "most common"
 - Bacterial: as streptococci, staphylococci, pneumococci.
 - Fungal: as candida albicans or cryptococcosis.
- 7- Neoplastic: e.g. lymphoma, leukemia.
- 8- Uremia
- 9- Familial Mediterranean fever.
- 10- Drugs e.g. hydralazine, procainamide and minoxidil.



Pathology

Dry pericarditis is associated with inflammatory cell infiltration and deposition of fibrin threads on pericardial surface along with formation of small amount of inflammatory exudate.

Clinical Picture

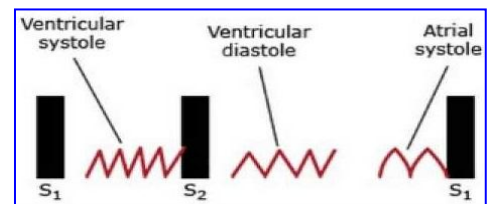
Symptoms:

1. May be asymptomatic.
2. Chest pain:
 - Cause: friction between the two layers of the pericardium
 - Character: stitching in nature.
 - Site: over the pericardium.
 - Increases by inspiration and movement of the trunk.
 - Relieved by leaning forward.
 - Referred to: the neck, epigastrium. Shoulders, the back.
3. Constitutional manifestations (fever, headache, malaise & anorexia) in inflammatory causes.
4. Symptoms of cause e.g. uremia.
5. Symptoms of complications e.g. Pericardial effusion.

Signs:

- Local:
 1. Pericardial rub:
 - Cause: friction between the two layers of the pericardium.
 - Nature: gritty sound.
 - Site: over the whole pericardium but best heard over the bare area and the base of the heart.
 - Timing: the sound has three components:
 - During atrial contraction.
 - During ventricular systole.
 - During ventricular diastole.

However one or two components may be heard.
 - Increases: by pressing stethoscope against chest wall.
 2. Signs of the cause e.g. myocardial infarction
 3. Signs of complications: e.g. pericardial effusion.
- General:
 - 1- Fever.
 - 2- Tachycardia and arrhythmia.
 - 3- Signs of cause e.g. uremia.
 - 4- Signs of complications: e.g. pericardial effusion.



Complications

- 1- Pericardial effusion.
- 2- Constrictive pericarditis.
- 3- Adhesive pericarditis.
- 4- Arrhythmia.

Investigations

ECG:

- Initially there is ST elevations in all leads (concave upwards) followed by ST depression and inversion of T-wave.
- Arrhythmia: particularly atrial e.g. AF.
- Evidence of cause: e.g. myocardial infarction.

Echocardiography:

- May show minimal collection of pericardial fluid.
- May show evidence of the cause e.g. myocardial infarction.

X-ray: No abnormality is detected.

Laboratory :

- Blood may show leucocytosis, elevated ESR. And C-reactive protein.
- Laboratory investigations for the cause e.g. elevated blood urea (uremia) or ANA(SLE).

Differential diagnosis

- 1- Other causes of chest pain e.g. myocardial infarction
- 2- Of the cause.

Treatment

- Treatment of the cause.
- Anti-inflammatory & analgesic drugs e.g. **Indomethacin**.
- Resistant cases may require steroids.

Pericardial effusion

Definition

Collection of fluid in pericardial sac.

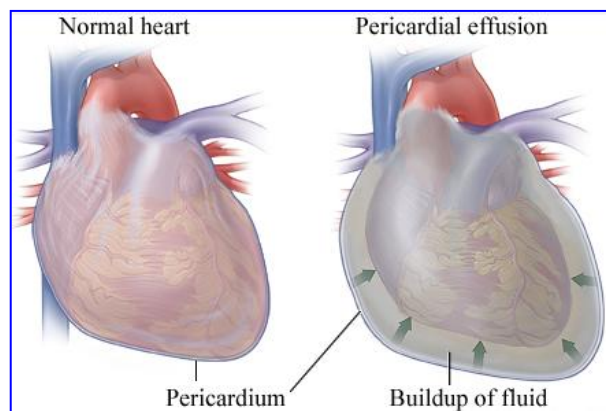
Etiology

1. Transudate (Hydropericardium)

- Congestive heart failure.
- Nephrotic syndrome.
- Liver cirrhosis.

2. Exudate (Seropericardium):

- All causes of dry pericarditis particularly:
 - T.B: Used to be the most common cause.
 - Idiopathic (probably viral) .
 - Rheumatic.
- Hemorrhagic pericarditis is a severe form of serous effusion containing many RBCs, and may occur in the following conditions:
 - Myocardial infarction
 - Uremia
 - TB
 - Malignant infiltration e.g. leukemia



3. Hemopericardium:

- Rupture heart as in trauma or myocardial infarction.
- Rupture of aortic aneurysm.
- Rupture coronaries during catheterization.
- Hemorrhagic blood diseases e.g. hemophilia, purpura.
- Over anticoagulants or thrombolytic therapy.

4. Chylous effusion:

- Rupture thoracic duct as in trauma or erosion by malignant tumor.
- Obstruction of thoracic duct by filariasis or tumors.

Pathology**1. Transudate:**

- Proteins: < 3 gm% or < 50 % of patient plasma proteins.
- Specific gravity: < 1015.
- LDH: < 200 IU/L or < 60 % of plasma LDH level.
- Cells : < 1000/cmm and usually only few epithelial cells.

2. Exudate:

- Proteins: > 3gm% or > 50 % of patient plasma proteins.
- Specific gravity: > 1015.
- LDH: > 200 IU/L or > 60 % of plasma LDH level.
- Cells: > 1000/cmm and usually inflammatory cells.

3. Hemopericardium: contains blood.**4. Chylous effusion:**

- Greasy, milky white with high fat content.
- Clears by addition of ether.
- Stains orange by Sudan III.

Hemodynamics

The effect of pericardial effusion depends on :

- The amount of fluid In pericardial sac.
- The rate of accumulation.

The presence of significant amount of fluid in pericardial sac (150 cc in acute cases and up to 2 liters in gradual cases) will lead to:

- Interfere with venous return leading to systemic congestion and low cardiac output.
- Pressure manifestations on the surrounding structures.

Clinical picture**Symptoms:**

1. Symptoms of systemic congestion: pain in then epigastrium and hypochondrium, dyspepsia, ascites followed by edema of LL (ascites precox).
2. Symptoms of low cardiac output.

3. Pericardial pain:

- Cause: stretch of pericardium.
- Nature: Dull aching pain.
- Site: Pericardial pain.
- Radiation: may be referred to the shoulders.

4. Pressure symptoms: occurs with massive effusion:

- Dyspnea: relieved by leaning forward (Mohammadian prayer position).
- Cough, dysphagia (Rare).

5. Symptoms of the cause e.g. uremia.

Signs:**➤ General:****I- Signs of systemic congestion:**

1- Congested neck veins:

- Pulsating neck veins except in severe cases, where neck veins appear non pulsating.
- Kussmel's sign: inspiratory filling of neck veins. This results from cardiac compression during inspiration resulting from traction on the pericardium by the diaphragm, and failure of the right atrium to accept the increased venous return during inspiration.
- Friedereich's sign (diastolic collapse): it is deep Y descent due to rapid emptying of the congested neck veins in a short time after opening of the tricuspid valve.
- Gibson's sign: deep X descent.

2- Enlarged tender liver

3- Ascites that may precede LL edema (ascites precox): This results from early hepatic congestion due to compression of hepatic veins at the their point of entry in the IVC by the pericardial infusion.

II- Signs of low CO:

1- Pulse:

- The pulse is weak and rapid.
- Pulsus paradoxus (decreased pulse volume during inspiration)
 - Cause: Normally during inspiration the expansion of lung leads to accommodation of more blood in the pulmonary circulation. However this is compensated by increase in the venous return to the right side. In pericardial effusion there s reduction in venous return during inspiration, with reduction in cardiac output → decrease in BP.
 - It can be detected by palpation especially in the femoral artery, or by the sphegnomanometer.
 - Other causes for Pulsus paradoxicus:
 - Constrictive pericarditis
 - Restrictive Cardiomyopathy
 - Severe bronchial asthma.

2- Low blood pressure with cold sweat.

III- Pressure signs:

- 1- Decubitus: May be Mohammadian prayer position.
- 2- Mediastinal syndrome may be present.

IV- Signs of cause.**➤ Local (cardiac signs) :**

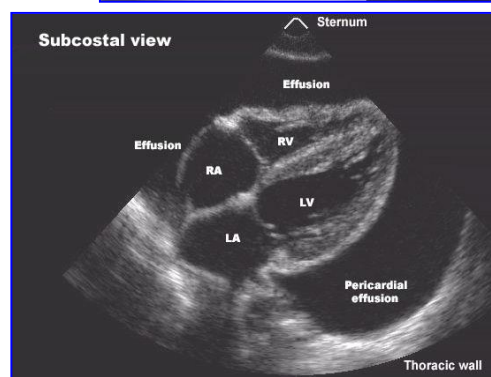
1. Inspection and palpation:
 - Pericardial bulge in children with long standing condition.
 - Apical pulsations is absent.
2. Percussion:
 - Dullness outside cardiac apex.
 - Enlarged bare area of the heart.
 - Dullness over the cardiac base which disappears by sitting (shifting dullness).
 - Dullness to the right border of the sternum.
 - *Ewart's sign*: Dullness over the left subscapular region due to lung collapse resulting from the compression of left lung by the pericardial fluid.
3. Auscultation:
 - absent or weak cardiac sounds with possible pericardial rub.

Investigations**1. Lab Studies**

- **CBC**: evidence of infection or cytopenias.
- **Cardiac enzymes** – To rule out myocardial ischemia.
- **for detection of the cause**:- RF, ANA, KFTs

2. Images:

- Chest X-ray:
 - Enlarged flask shape cardiac shadow.
 - Stenciled cardiac borders: smooth cardiac borders with no angulations
 - Double contour of cardiac border.
 - Change of cardiac shape with change in patient position.
 - Change in cardiac shape from day to day.
 - *Rotch's sign*: Obtuse cardiac phrenic angle due to left lower lobe collapse.
 - Pulmonary oligemia.
 - Diminished cardiac pulsations under screen.
- ECG:
 - Low voltage.
 - Flat or inverted T wave.
- Echocardiography:
 - Detects pericardial effusion.
 - Determines the severity (cardiac tamponade causes diastolic collapse of RV and the free wall of RV).
- Cardiac catheterization: Not required .



- CT scan : may detect as little as 50 CC of fluids.
- MRI : can detect as little as 30 cc of pericardial fluid. And may be able to distinguish hemorrhagic and non hemorrhagic.
- Transesophageal echocardiography (TEE) : is useful in loculated effusion.

3. **Aspiration of effusion:**

- **Indication:**
 - **Diagnostic:** may allow detection of cause by determining the nature of the pericardial fluid:
 - ✚ **Transudate:** low protein content (< 3gm % or less than 50% of plasma proteins, low specific gravity (<1015) and low cell number (< 1000/cmm).
 - ✚ **Exudate:** high protein content (> 3gm % or more than 50% of plasma proteins, low specific gravity (>1015) and high cell number (> 1000/cmm).
 - ✚ **Hemopericardium:** contains RBCs and high LDH
 - ✚ **Chylous:** whitish, greasy, high fat content, clears by ether and stains black by Sudan III.
 - **Therapeutic:** to reduce the intrapericardial pressure.
- **Site of aspiration:**
 - The angle between the xiphoid process and the left costal margin (the commonest site).
 - Outside the cardiac apex .
 - The bare area of the heart.
- **Method of monitoring:**
 - Echocardiography
 - ECG.

Differential diagnosis:

1. Constrictive pericarditis.
2. Right-sided heart failure.
3. Restrictive cardiomyopathy.
4. Tricuspid stenosis.
5. Liver cirrhosis & nephritic syndrome.

Treatment

A. Medical

- Treatment of the cause: e.g. in tuberculous effusion.
- Steroids may be added to reduce inflammation & prevent constrictive pericarditis.

B. Pericardial aspiration: (Pericardiocentesis):

- In cardiac tamponade.
- May be used in suppurative effusion for drainage of pus & injection of antibiotics.
- May be used in (malignant effusion for injection of cytotoxic drugs).

C. Pericardial drainage:

1. *Subxiphoid pericardial window with pericardiostomy.*
2. *Thoracotomy* (Pleuropericardial window).
3. *Video-assisted thoracic surgery (VATS).*
4. *Balloon pericardotomy* by catheterization.

D. Pericardial sclerosis (Pericardiocentesis)

In **resistant cases** by using sclerosing agents (e.g., tetracycline, doxycycline, cisplatin)

Complications include intense pain, atrial arrhythmias and infection.

E. Pericardiectomy:

Indicated in cases of massive & recurrent effusions not responding to medical treatment.

Constrictive pericarditis "PICKS DISEASE"

Definition

Fibrous adhesions between the two layers of the pericardium.

Etiology

- The same as dry pericarditis with **exception of rheumatic fever.**
- **Tuberculosis** is the most common.

Pathology

Fibrous adhesions between the parietal and visceral pericardial layers with limitation in cardiac expansion and relaxation.

Clinical picture**Symptoms:**

1. Symptoms of systemic congestion: pain in then epigastrium and hypochondrium, dyspepsia, ascites followed by edema of LL(ascites precox).
2. Symptoms of low cardiac output.
3. No Pericordial pain.
4. No pressure symptoms.
5. Symptoms of the cause.

Signs:➤ **General:****I-Signs of systemic congestion:**

1. Congested neck veins:
 - Pulsating neck veins except in severe cases were neck veins appear nonpulsating
 - Kussmel's sign
 - Friedereich's sign (diastolic collapse)
 - Gibson's sign: deep X descent
2. Enlarged tender liver
3. Ascites that may precede LL edema (ascites precox).

II- Signs of low CO:1. Pulse:

- The pulse is weak and rapid.
- Pulsus paradoxicus (decreased pulse volume during inspiration).

2. Low blood pressure with cold sweat.**III- Signs of cause.****➤ Local (cardiac signs) :**1. Inspection and palpation:

- No cardiac enlargement
- Systolic retraction of the pericardium .
- Apical pulsations is weak or absent.

2. Auscultation:

- Weak distant cardiac sounds.
- S3 : sudden halting of the relaxing ventricles by the pericardium (Pericardial knock) .

Investigations

- Chest X-ray:
 - Calcified pericardium may be present especially in cases with TB
 - The heart is small.
 - Pulmonary oligemia.
 - Diminished cardiac pulsations under screen.
- ECG:
 - Low voltage.
 - Flat or inverted T wave.
- Echocardiography:
 - Excludes the presence of pericardial effusion.
 - May show pericardial thickening or calcification.
- CT & MRI: may be used to verify diagnosis in some cases.
- Cardiac catheterization: may show square root sign in pressure tracing of the RV and LV (diastolic dip followed by plateau).

**Differential diagnosis**

1. Pericardial effusion.
2. Right-sided heart failure.
3. Restrictive cardiomyopathy.
4. Tricuspid stenosis.
5. Liver cirrhosis & nephritic syndrome.

Treatment

- Pericardiectomy with treatment of the cause.
- Symptomatic treatment; e.g. for oedema & ascites.

Adhesive pericarditis

Definition

Fibrous adhesions between the pericardium and the surrounding mediastinal structures & chest wall.

Etiology

It is rare late complication of rheumatic fever.

Pathology

There is adhesion between the parietal pericardium and the surrounding mediastinal structures.

Clinical Picture

- Clinical picture of associated valvular affection.
- Fixed apex: the apex does not change in site with change in patient's position.
- *Broadbent's sign*: due to adhesions, there is successive retraction of the lower sternum and posterior intercostal spaces.

Investigations

- Evidence of associated valve lesion by ECG, X-ray and echocardiography.
- Under screen there is evidence of esophageal kinking with every cardiac contraction.

Treatment

Treatment of the underlying heart disease.

Myocarditis

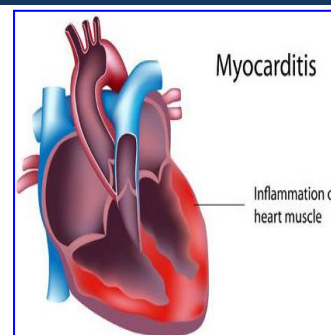
Definition

inflammation of myocardium.

Aetiology (4Is)

Infections

- Viral (adenovirus, parvovirus, HIV, enterovirus, rubella, polio, CMV)
 - Protozoan (Chagas disease and Toxoplasma)
 - Bacterial (Brucella, diphtheriae, gonococcus, H.influenzae, Rickettsia)
 - Fungal (Aspergillus)
 - Parasitic (ascaris, schistosoma, Taeniasolium, Trichinellasprialis)
- Bacterial myocarditis is rare in patients without immunodeficiency*



Iatrogenic : Drugs (ethanol and some forms of chemotherapy)

Immunological

- Allergic drug reaction (acetazolamide).
- Rejection after a heart transplant.
- Collagen (scleroderma, SLE, sarcoidosis, systemic vasculitis such as Churg-Strauss syndrome, and Wegener's granulomatosis).
- Toxins (arsenic, toxic shock syndrome or snake venom).

Irradiation: (Physical agents) :- Electric shock, hyperpyrexia.

Clinical picture

- Features of the cause : e.g. viral infection.
- Chest pain.
- Congestive heart failure.
- Palpitations (due to arrhythmias).
- Sudden death (in 20% of cases) .
- Myocarditis is often associated with pericarditis.

Investigations

ECG:-	- Tachycardia - Non specific changes - Features of complications:- IHDs or pericarditis changes
Echo & doppler	cardiomegaly and contractile dysfunction
MRI	Diagnostic
Myocardial biopsy:- the most diagnostic but invasive Investigation for detection of the cause e.g. serology of viruses	

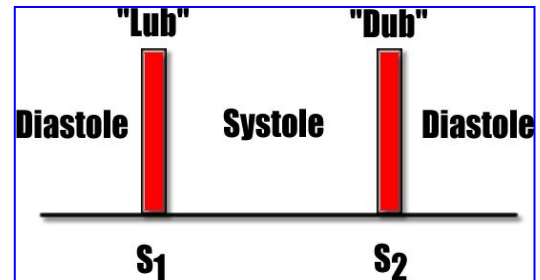
Treatment (3Ss)

- 1- Symptomatic treatment: analgesics and antipyretics
- 2- Supportive :-
 - anti heart failure.
 - control of arrhythmia.
- 2- Steroids and immunosuppressive drugs.

Heart sounds

First heart sound

- Cause: closure of mitral and tricuspid valve.
- Timing: beginning of systole.
- Increases in: MS, TS, and tachycardia.
- Decreases in: TR, MR, bradycardia.
- Site of comment: Apex and tricuspid areas.

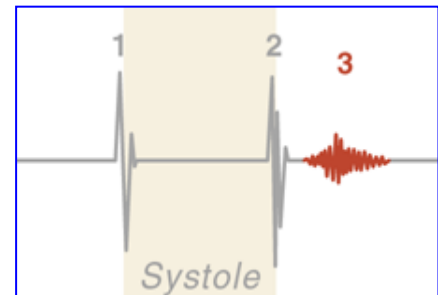


Second heart sound:

- Cause: closure of aortic and pulmonary valves.
- Timing: Beginning of diastole.
- Increased in systemic and pulmonary hypertension.
- Decreased in AS, AR, PS, PR.
- Site of comment: in aortic and pulmonary areas.
- Splitting:
 - Increased in PS, ASD, RBBB.
 - Decreased in AS, PDA, LBBB.

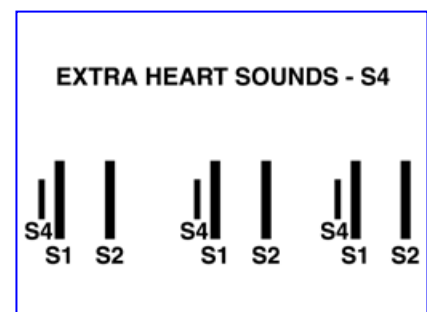
Third heart sound

- Cause:
 - Increased blood flow from atrium to ventricle.
 - Flabby myocardium as HF, or ischemic heart.
- Timing: in diastole, isolated from 2nd heart sound by isometric relaxation phase.
- Site of comment: at apex or tricuspid area.



Fourth heart sound

- Cause: powerful atrial contraction.
- Timing: Presystolic, before 1st heart sound.
- Site of comment at apex or tricuspid area.



Valvular heart diseases

In any valve lesion

Aetiology

	Stenosis	Regurge
Usual	- Rheumatic fever. - Congenital	
Spec.	Calcific	- Infective endocarditis - Surgical
Relative	- Relative:- increased B flow	- Functional (Dilatation of the ring)

Acute valve Regurge :- Surgical - infective endocarditis + (IHDs in MR-TR)

Clinical picture (C/O - Signs - Complications)

C/O:

- Aorta = Chest pain
- Regurge = palpitation (NB: AF = Irrg. palpitation)
- MS = 1st P. congestion

Signs

A- General:-

- Pulse - ABP
- Peripheral sign = AR
- NVs = Right sided (PH++ - TR - RVF)
- Signs of
 - P. congestion=BBC
 - S. congestion
 - Low COP

B- Cardiac:

Precordial examination (inspection-palpation-percussion):

- Chamber +++
- Apex
- Thrill

Auscultation: you must mention over which area

- HS
- Add. S
- Murmur 5 (time-chr-site- propagation-how to increase)

Complications

- 1- HF 2- Infective endocarditis 3- Rheumatic activity

Investigation

X-ray	ECG	Echo (the most diagnostic)	Catheterization
- Chamber ++ - P. vasculature - Calcific Valve	- Chamber ++ - Arrhythmia - IHDs	Aetiology - lesion Function (EF) - Complications - Severity ?????	As Echo + therapeutic

Treatment

- Medical - Interventional - Surgical

Mitral stenosis (MS)

Aetiology

1. Rheumatic fever:- (the most common cause).
2. Congenital: a rare cause.
3. Relative stenosis (not an organic stenosis)
 - Increased blood flow through the mitral valve.
 - Carey-coomb's murmur in acute rheumatic valvulitis.
 - Austin flint murmur in severe aortic regurgitation.

Pathophysiology

- In mild cases:
The blood flow through the mitral valve remains normal.
No symptoms occur.
- In severe cases: (valve area less than 2 cm^2): Decreased blood flow.
Blood stagnate in pulmonary veins (**pulmonary venous congestion**).
- Later on:
Vasoconstriction of pulmonary arterioles --- ↓ P. congestion + **Pulmonary hypertension**.
- Finally:
RV enlargement & then RVF will occur secondary to pulmonary HTN.

Therefore four stages will occur in patient with MS

Stages of Mitral stenosis

- **Stage one:** "Mild or asymptomatic mitral stenosis"
The only abnormality is anatomical narrowing of the mitral valve, but no symptoms.
- **Stage two:** "mitral stenosis with pulmonary congestion".
- **Stage three:** "mitral stenosis with pulmonary hypertension"
- **Stage four:** "mitral stenosis with right ventricular failure"

Clinical picture

There is a **latent period** of several years between the initial attack of rheumatic carditis & the development of manifestations of mitral stenosis.

Symptoms Does the patient with MS always symptomatize???

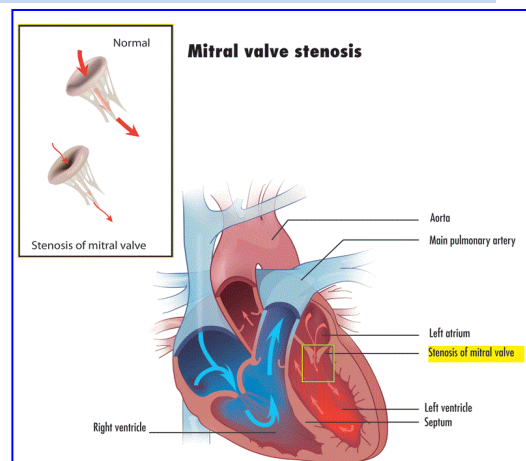
Stage one:	No symptoms.
Stage two:	Symptoms of pulmonary congestion (but pulmonary oedema is not common). Symptoms of low CO.
Stage three:	Symptoms of pulmonary congestions: improve. Symptoms of Low CO: increase.
Stage four:	Symptoms of systemic congestion

Dyspnea is the most common symptom

Signs

General:

Stage one:	No signs.
Stage two:	signs of pulmonary congestion .
Stage three:	signs of low cardiac output .
Stage four:	signs of systemic congestion .



Cardiac:**A. Precordial examination:**

Stage one:	Apex: normal site & slapping character.
Stage two:	Diastolic thrill: ending in a palpable S1.
Stage three:	The previous findings.
Stage four:	+ Signs of: Pulmonary hypertension. Signs of: RV enlargement.

B. Auscultation (Summary)

Stage one:	Over Mitral area
Stage two:	<u>HS:-</u> * Accentuated S1 <u>Add. Sound:-</u> *Opening snap <u>Murmur:-</u> Mid-diastolic with presystolic accentuation - rumbling - increased on left lateral position
Stage three:	Over Pulmonary area <u>HS:-</u> Accentuated S2 <u>Add. Sound:-</u> Ejection click + S4 (Over T area) <u>Murmur:-</u> Systolic ejection murmur. - Soft early diastolic murmur: (Graham Steel murmur)
Stage four:	As in RV failure:- Over Tricuspid area S3 gallop + Functional TR pansystolic murmur

Stage one & stage two (over the mitral area)1. Accentuated first heart sound: due to:

- + Fibrosis of the mitral cusps.
- + Forcible closure of the mitral cusps: because:
 - ❖ They are displaced downwards due to high LA pressure.
 - ❖ The mitral valve is opened as wide as possible during the diastole.

N.B. Diminished S1 in mitral stenosis denotes:

- Double mitral lesion (with predominant regurge) - Calcified mitral valve.

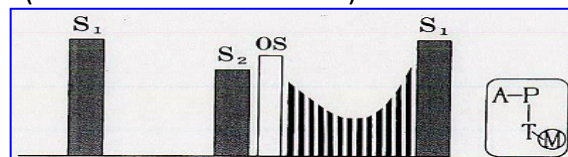
2. Mitral opening snap:

- It is **sharp & snapping** due to opening of the rigid cusps of mitral valve.
- It heard in the **early diastole**.
separated from S2 by the isometric relaxation phase - Just before the murmur.

Its Significance:- Sure MS - **non-calcific** valve - severity

3. Murmur of mitral stenosis:

- Timing:
 - * Mid-diastolic pre-systolic with pre-systolic accentuation.
 - * In AF, there is loss of pre-systolic accentuation (loss of atrial contraction).
- Character: rumbling.
- Site: at or slightly inside the apex.
- Propagation: not propagated.
- Position:
 - * Best heard with the cone of the stethoscope.
 - * In the left lateral position.



Silent mitral stenosis: (MS with no murmur) due to:

- 1- High LV pressure: LVF.
- 2- Low LA pressure (- Severe pulmonary HTN. - RVF).
- 3- In association with ASD.

Stage three

- The previous findings.
- Auscultatory findings of pulmonary hypertension:
 - a. Over the pulmonary area:
 - S2 is accentuated.
 - Systolic ejection click.
 - Systolic ejection murmur.
 - Soft early diastolic murmur: (Graham Steel murmur)
 - b. Over the tricuspid area:
 - S4 gallop

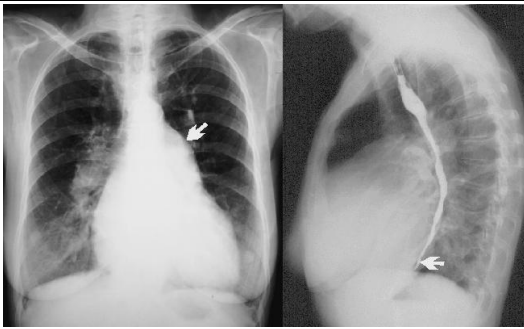
Stage four

- The previous findings.
- Auscultation over tricuspid area:
 - Pan systolic murmur of functional tricuspid regurge.
 - Proto-diastolic gallop (S3) due to RVF.

Complications

1. In the mitral valve:
 - Rheumatic activity.
 - Calcification.
 - Infective endocarditis.
2. In the left atrium:
 - a) Arrhythmias, especially AF.
 - b) LA enlargement, causing symptoms:
 - On esophagus: dysphagia.
 - On left bronchus: dyspnea & cough.
 - On left recurrent laryngeal nerve: hoarseness of voice.
 - c) Thrombo-embolic complications:
 - Systemic embolization: e.g. Cerebral, peripheral, renal.
 - Ball & valve embolus: leading to syncope & sudden death.
3. In the right ventricle: RVF.
4. In the left ventricle: No LVF in isolated mitral stenosis.
5. In the lung:
 - Hemoptysis.
 - Pulmonary infection.
 - Pulmonary embolism (secondary to DVT0.
 - Pulmonary oedema is not common in mitral stenosis.
6. Complications of surgery.

Investigation:-

X- Ray	Stage one: no abnormality. Stage two: Mitralization (LA++) - Obliteration of the waist - Double contour of the right border - Pulmonary congestion Stage three & four: - Right ventricular enlargement. - Pulmonary hypertension. - May be calcified mitral valve.	
ECG	Stage one: no abnormality. Stage two: LA +++ (P mitrale: broad & bifid). Stage three & four: RA +++ (P pulmonale: tall & peaked). + RV+++	
Echo	The most sensitive & specific non-invasive method diagnosing MS. - Detects the severity (Valve area < 1 cm²) - Chamber enlargement - function (EF)	
Catheter	AS ECHO	

Ba Swallow: Enlarged left atrium displaces the esophagus posteriorly.

Tight MS is diagnosed:

- **According to the stage:**
 - Stage two with dyspnea more than grade two, or
 - Stage three, or Stage four.
- **According to the echocardiography:**
 - Valve area less than 1 cm².

Treatment**1. Medical:**

- a) Prophylaxis against: rheumatic activity, infective endocarditis (uncommon).
- b) Symptomatic for: complications **e.g.** HF, AF, infection, embolization.

2. Surgical:

- a) **Indications:**
 - Tight mitral stenosis (valve area is < 1 cm²).
 - Marked symptoms not responding to adequate medical treatment.
 - Embolization with no serious deterioration of the condition of the patient.
- b) **Types of operations:**
 - i. Mitral commissurotomy: closed or open (Old regimen of limited indications)
 - ii. Valve replacement: By a prosthesis (tissue or synthetic).
 - in - Calcific valve - Associated mitral regurge. - Failed commissurotomy.
- c) **Complications:-**
 1. Embolization.
 2. Arrhythmias.

3. Mitral incompetence.
4. Restenosis.
5. Post-cardiotomy syndrome: Pleuro-pericarditis 10 – 15 days post-operative as an allergic process due to injury of the pericardium during surgery.
6. Complications of artificial valves:
 - Infective endocarditis.
 - Thrombo-embolism.
 - Mechanical dysfunction.
 - Hemolytic anemia.

3. Interventional:- Balloon dilatation:

- May be performed in some patients indicated for valvotomy.

Mitral regurgitation (MR)

Aetiology

A. Organic:

1. Rheumatic fever: the most common cause.
2. Congenital.
3. Prolapse of the mitral valve (MVP)
4. Papillary muscle dysfunction **e.g.** CAD.
5. Infective endocarditis.
6. Iatrogenic: following mitral valvotomy.

Acute MR:-

- 1- post-valvotomy
- 2- Severe inf endocarditis (perforation)
- 3- acute papillary Ms. rupture (MI)

C/p:- presented with APO

- #### B. Relative:-
- Dilatation of the mitral ring secondary to LV dilatation.

Pathophysiology

1. During systole: Part of blood regurgitates from LV to LA leading to:
 - Low CO.
 - LA dilatation (blood from 2 sources)
2. During diastole:-
A large volume of blood → LV → LV enlargement which may end in LVF.

Clinical picture

Symptoms

1. No symptoms: in early cases.
2. Symptoms of low cardiac output: in late cases.
3. Symptoms of pulmonary congestion: in late cases.
4. **PALPITATION.**

Palpitation is the most common

Signs

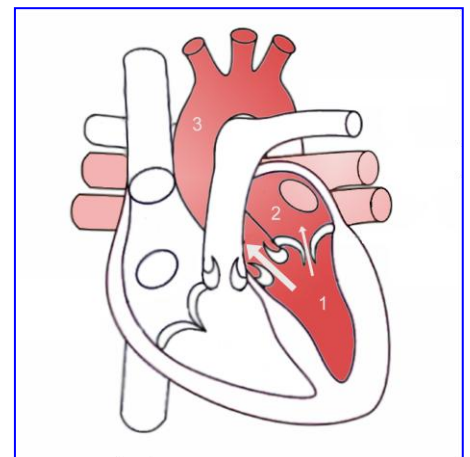
General:

- * No signs: in early cases.
- * Signs of low cardiac output: in late cases.
- * Signs of pulmonary congestion: in late cases.

Cardiac:

A. Precordial examination:

- * Signs of LV enlargement: with hyperdynamic apex.
- * Systolic thrill: over the apex.



B. Auscultation:

HS	Weak S1 (muffled) due to failure of proper mitral closures
Add S	S3 (excessive flow of blood from LA to LV)
Murmur	<u>Timing</u> : pansystolic starting with S1. <u>Character</u> : soft or harsh. <u>Site</u> : over the apex. <u>Propagation</u> : - To the axilla - - To the base of the heart & medially in posterior leaflet disease. <u>Position</u> : heard best in the left lateral position

Complications

Same complications of MS, but:

1. Infective endocarditis: is common.
2. Left ventricular failure: occurs.

Investigations

X- Ray	* No abnormality: in early cases. * Enlarged LA & LV: in late cases. * Pulmonary congestion: in late cases. * Calcified mitral valve especially: in double mitral lesion.
ECG	* Enlarged LA (P mitrale:- broad & bifid). * Enlarged LV.
Echo	* Detects the severity of mitral regurgitation. * Detects chamber enlargement. * Detects the cause: e.g. MVP .
Catheter	AS ECHO

Treatment

1. Medical: Same as that of mitral stenosis.

2. Surgical: Valve replacement.

Mitral valve prolapse (Barlow's syndrome – Floppy mitral valve)

- It is common in young females.
- Mild prolapse is regarded as a normal variant.

Aetiology

1. **Unknown**, but there is familial incidence.
2. Isolated abnormality, **but** may be associated with: **Marfan's syndrome or ASD**.

Pathophysiology

During systole, a mitral valve leaflet (commonly the posterior) prolapses into the L.A. This may result in papillary muscle strain & some mitral regurge.

Usually the case is not hemodynamically significant.

Clinical pictures

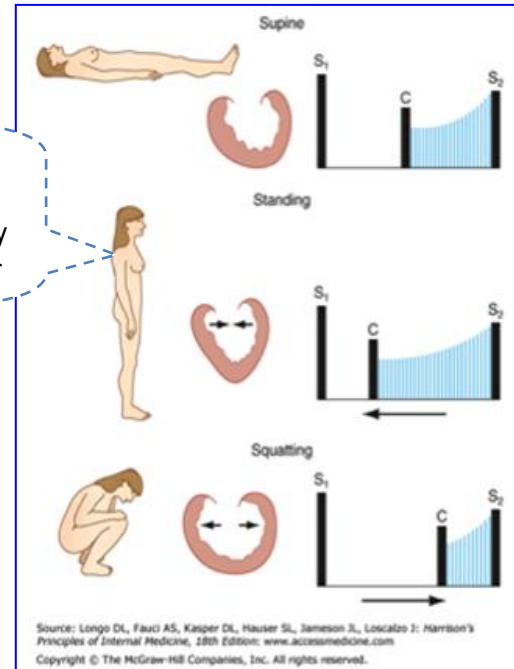
Symptoms

- * Atypical chest pain: is the most common symptom.
- * Palpitation.

Signs

- * Mid systolic click.
- * Late systolic murmur.

In contrast to most other heart murmurs, the murmur of mitral valve prolapse is accentuated by standing and valsalva maneuver



Complications

- * Progressive mitral regurgitation.
- * **Infective endocarditis.**
- * Systemic embolization.
- * Arrhythmias.
- * Sudden death.

Investigations

Echocardiography is diagnostic.

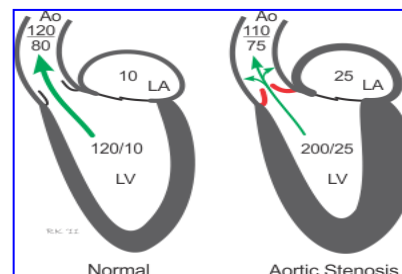
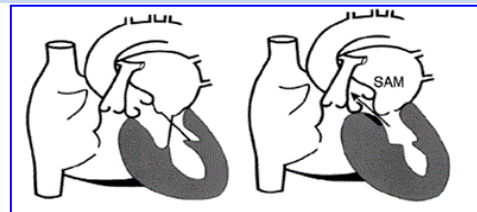
Treatment

- * Reassurance.
- * Prophylaxis against: **infective endocarditis.**
- * Anti-arrhythmic therapy.
- * **Propranolol:** is effective for chest pain.
- * Surgery: mitral valve replacement in cases with severe MR

Aortic stenosis (AS)

Aetiology

1. Rheumatic fever.
2. Calcific.
3. Congenital:
it may be: valvular, subvalvular or supravalvular.
4. Hypertrophic cardiomyopathy (Idiopathic Hypertrophic Subaortic Stenosis; IHSS):
It produces a subvalvular obstruction in the systole due to: contraction of the hypertrophied interventricular septum.
5. Relative stenosis (not an organic stenosis):
 - a. Dilatation of the aorta:
Hypertension, atherosclerosis, aortic aneurysm.
 - b. Increased blood flow across the aortic valve:
Hyperdynamic circulation, AR.



Pathophysiology

- During systole, there is obstruction of blood flow from LV to aorta leading to:
 1. Low cardiac output.
 2. Pressure overload on LV leading to LVH & LVF.
- Normally, the aortic valve area is 3 – 4 cm². In severe AS, it is less than 0.8 cm².

Clinical picture

Symptoms

1. No symptoms: in mild cases.
2. Symptoms of low CO: in severe cases.
3. Symptoms of pulmonary congestion: due to LVF.
4. SYNCOPE: especially exertional due to low fixed CO.
5. ANGINA: due to
 - Reduced coronary blood flow: due to low CO & shortened diastole.
 - Left ventricular hypertrophy: increases the myocardial O₂ demands.
 - Associated coronary atherosclerosis: especially in calcific AS.

Signs

General:

1. Pulse:
 - * Pulsus parvus et tardus (plateau pulse): *rises slowly, of small volume, returns slowly.*
 - * Pulsus bisferiens: *bifid pulse occurring in double aortic lesion.*
2. BP: low SBP in severe cases.
3. Systolic thrill: over the carotid arteries.

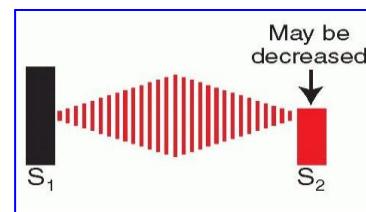
Cardiac:

A. Precordial examination:

1. Signs of LVH: with a heaving apex.
2. Systolic thrill: over second right space.

B. Auscultation:Over the aortic area:

1. Second heart sound: weak.
2. Systolic ejection click: due to opening of the rigid cusps.
3. Systolic ejection murmur:
 - Midsystolic, harsh.
 - Maximum over second right space → apex & carotid arteries.

Over the pulmonary area:

- Reversed splitting of the second heart sound.

Over the mitral areas:

1. S4.
2. Propagated murmur of AS.

complications

1. LVF.
2. Infective endocarditis.
3. Sudden death: usually due to VF.
4. Heart block: in calcific AS due to extension of calcification to AV bundle.
5. Rheumatic activity: in rheumatic AS.

Investigations

X- Ray	No abnormality: in mild cases. LV: LVH. Lungs: pulmonary congestion when LVF occurs. Aorta: Small aortic knuckle <u>or</u> post-stenotic dilatation. Aortic valve calcification may be seen.	
ECG	* Enlarged LV.	
Echo	* Detects the severity of stenosis by: - Measurement of valve area. - Measurement of pressure gradient across the valve. * Detects the type of stenosis. * Detects LVH	
Catheter	AS ECHO	

Tight AS:-

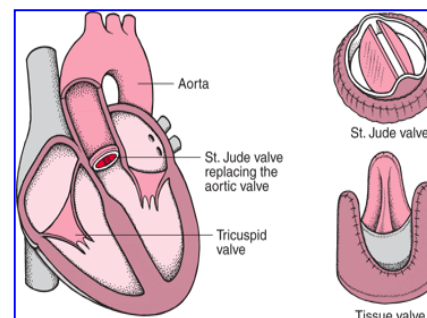
- 1- Pressure gradient > 50 mmHg
- 2- Valve area > 0.8 cm²

Treatment**1. Medical:**

- Same as that of mitral stenosis.
- Anginal attacks: may be relieved by SL nitrates.

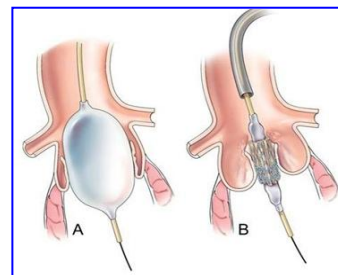
2. Surgical: (Aortic valve replacement) indications:

- a. Presence of severe symptoms.
- b. Pressure gradient more than 50 mmHg.
- c. Valve area less than 0.8 cm².



3. Balloon dilatation: *indications:*

- a. **Children:** with congenital AS as an alternative to surgery.
- b. **Elderly:** with severe calcific AS who are too ill to undergo surgery.

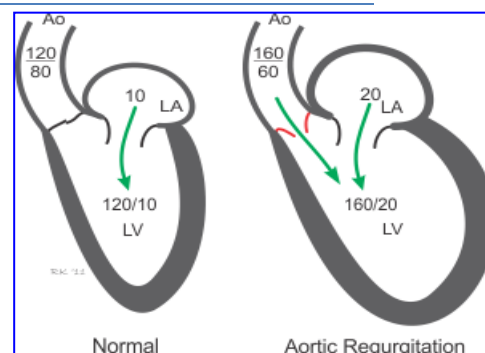


	Congenital	Rheumatic	Calcific
Age	Young	Young & middle	Old
History of RF	Absent	Present	Absent
Type	Valvular or sub/supravalvular	Valvular	Valvular
Associations	Congenital anomalies	Other valvular lesions	Atherosclerosis

Aortic regurgitation (AR)

Aetiology

1. Rheumatic fever: **the most common cause.**
2. Infective endocarditis.
3. Congenital.
4. Dilatation of the ascending aorta as in :
 - a. Syphilitic aortitis (Dilated aortic ring).
 - b. Severe hypertension.
 - c. Ankylosing spondylitis.
 - d. Aortic aneurysm: with dissection.
 - e. Marfan syndrome.



Pathophysiology

Regurgitation of blood from the aorta to the LV in diastole:

1. Increased LV stroke volume, this results in:
 - a. **Increased SBP.**
 - b. Peripheral VD which (together with regurgitation) will **decrease the DBP.**
 - c. **↑ SBP & ↓ DBP → wide pulse pressure** causing peripheral signs of AR.
2. Volume overload on the LV leading to LV dilatation & later on LVE.

Clinical picture

Symptoms

1. Generalized **body throbbing:** due to increased arterial pulsation.
2. **Palpitation:** due to forcible LV contraction.
3. Symptoms of pulmonary congestion: when LVE occurs.
4. Angina pectoris: "two types of angina occur in aortic regurge"
 - **Classic angina of effort** : Decreased DBP: reduces coronary filling.
 - **Angina of Lewis:**
Nocturnal & associated with autonomic disturbance **e.g.** sweating & tachycardia.

Signs**General: "Peripheral signs of aortic regurg"**

1. De-Musset sign: nodding of the head.
2. Corrigan's sign: prominent carotid pulsations.
3. Systolic thrill: over the carotid arteries.
4. Water hammer pulse: rises rapidly, of big volume, collapses rapidly.
5. Capillary pulsations: detected in nail bed, lips or ear lobule.
6. Pistol shots: loud booming sounds heard with each pulse beat over the arteries (especially femoral) due to sudden distension of collapsed arteries.
7. Duroziez's sign:
 - Systolic & diastolic murmurs over the femoral artery after be compressed with the stethoscope bell.
 - The systolic murmur is due to the rapid flow of blood to periphery
 - The diastolic murmur is due to rapid regurge of blood to the heart.
8. Blood pressure:
 - a. Wide pulse pressure:
 - Exaggerated difference between systolic & diastolic blood pressure.
 - b. Hill's sign:
 - Exaggerated difference between SBP in LLs & ULs: more than 50 mmHg.
 - Normally, SBP in LLs is higher than in ULs: by about 10 – 20 mmHg.

Cardiac

- A. Precordial examination:
 - Signs of LV enlargement: with hyperdynamic apex.
 - No thrill over the aortic area: in isolated AR.
- B. Auscultation:
 - Over the aortic area:
 - a. Normal second heart sound.
 - b. Murmur of AR:
 - Timing: early diastolic.
 - Character: soft blowing, decrescendo.
 - Site: maximum over the third space.
 - Propagation: to the apex.
 - Position: **best heard with the diaphragm of the stethoscope, the patient is:**
 - Sitting up. - Leaning forward. - Holding breath in forced expiration.
 - c. Soft ejection systolic murmur:
 - Due to ↑ blood flow across the aortic valve (relative AS).
 - Over the mitral area:
 - a. S3.
 - b. **Propagated murmur: of AR.**
 - c. Pan systolic murmur: of functional MR.
 - d. **Austin Flint murmur**: (mid-diastolic) due to:
 - Elevation of anterior leaflet of the mitral valve by the regurgitant blood from the aorta causes relative **MS**.



Complications

1. Rheumatic activity.
2. Infective endocarditis.
3. LVF.

Investigations

X- Ray	"Aortic configuration (Boot-shaped heart)" - LV enlargement & dilated aorta.
ECG	* LV++
Echo	• Detects the severity of the lesion. • Detects chamber enlargement.
Catheter	AS ECHO

**Treatment****1. Medical:**

- Same as that of mitral stenosis.
- Syphilis: anti-syphilitic treatment.

2. Surgical: (aortic valve replacement) *indications:*

- a. Presence of severe symptoms.
- b. Progressive cardiomegaly.
- c. Declining LV functions.

Pulmonary stenosis (PS)**A. Organic:**

- Congenital: the most common cause.
- Carcinoid syndrome: rare.

B. Functional:

- Pulmonary hypertension.

Pulmonary regurgitation (PR)**A. Functional:**

- Mostly due to **PH** which causes dilatation of the pulmonary ring.

B. Organic:

- Congenital.
- Carcinoid syndrome.

Tricuspid stenosis (TS)

Aetiology

- Rheumatic fever: the most common cause, & *is almost always associated with M.*
- Carcinoid syndrome: a rare cause.

Pathophysiology

During diastole, there is obstruction of blood flow from RA to RV leading to:

- Systemic venous congestion.
- Low CO.

Clinical picture

Symptoms

1. Systemic venous congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - Congested pulsating neck veins with giant A wave.
 - Enlarged tender pulsating liver.
 - Ascites **before** oedema of lower limbs (*ascites precox*).
2. Low CO.

Cardiac:

- A. Precordial examination:
 - RA enlargement (dullness to the right border of the sternum).
 - RV enlargement (due to frequently associated MS).
- B. Auscultation: *Over the tricuspid area*:
 - Accentuated first heart sound.
 - Opening snap.
 - Mid-diastolic murmur that increases by inspiration.

Investigations

X- Ray	- RA & RV enlargement
ECG	- RA & RV enlargement
Echo	• Detects the severity of the lesion. • Detects chamber enlargement.
Catheter	AS ECHO

Treatment

1. **Medical:** Treatment of right sided heart failure.
2. **Surgical:** Valve replacement.

Tricuspid regurgitation (TR)

Aetiology

1. Functional: **the most common cause** due to dilatation of the tricuspid ring secondary to RV dilatation as **M.S.**
2. Organic: **rare:**
 - Rheumatic fever.
 - Infective endocarditis.
 - Congenital.
 - Carcinoid syndrome.

Pathophysiology

During systole, blood regurgitates from RV to RA causing **(V. overload)**

- Low CO.

+ - RA enlargement. - RV enlargement. - RVF (S. congestion).

Clinical picture

Symptoms

1. Systemic congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - *Congested pulsating neck veins: with systolic expansion.*
 - *Enlarged tender pulsating liver.*
 - *Ascites **before** oedema of LLs (Ascites precox).*
 - *Mild jaundice & peripheral cyanosis (cyano-icteric face).*
2. Low CO.

Cardiac:

- A. Precordial examination:
 - RA & RV enlargement.
 - Rarely: systolic thrill over tricuspid area.
- B. Auscultation: "over tricuspid area"
 - a. Weak muffled S1.
 - b. S3.
 - c. Pan systolic murmur.
 - Timing: pan systolic.
 - Character: soft or harsh.
 - Site: tricuspid area.
 - Propagation: to the apex, but not to the axilla.
 - Caravallo's sign: murmur increases with inspiration being of right side origin.

Investigations

X- Ray	- RA & RV enlargement
ECG	- RA & RV enlargement
Echo	• Detects the severity of the lesion. • Detects chamber enlargement.
Catheter	AS ECHO

Treatment

1. **Medical:**
 - Treatment of right sided heart failure.
2. **Surgical:**
 - Valve replacement.

The heart with multivalvular lesions what to do??

First

Valuable information may be observed in JVP and in arterial pulse:

- Raised JVP with prominent systolic wave = tricuspid regurgitation.
- Corrigan's sign – waterhammer = aortic regurgitation.
- Pulsus bisferiens (double strokes): aortic stenosis + aortic regurgitation.

Second

Pick the very significant abnormal signs suggestive of one lesion and then confirm by auscultation *i.e. one lesion by one.*

1. **Hyperdynamic apex:** either aortic or mitral regurgitation:
 - **Aortic regurgitation:** after noting Corrigan's signs (vigorous pulsation of the carotids) – waterhammer pulse: big pulse pressure with very low diastolic < 60 mm. Then confirm by listening carefully to soft blowing early diastolic murmur at the lower right parasternal area.
 - **Mitral regurgitation:** check by feeling a **systolic thrill** at the apex (patient lying on his left side after exercise)
Confirm by listening to pansystolic murmur maximal over the mitral.
A mid diastolic murmur may also be heard (mitral stenosis).
2. **Signs of pulmonary hypertension:**
 - Left parasternal heave. (RV +++).
 - Pulsations and dullness in second left space.
 - Diastolic shock. (accentuated P2).
 - Prominent "a" wave.

Practically mitral stenosis is highly suspected.

- Confirm then by listening carefully to a mid diastolic murmur commonly with a presystolic accentuation.
 - Otherwise suspect other causes of pulmonary hypertension.
3. **Heaving apex:** aortic stenosis or severe hypertension.
Aortic stenosis check with feeling a systolic thrill: patient sitting and leaning forwards, holding his breath in expiration.

Confirm by listening to a harsh ejection murmur over aortic area propagated to the root of neck.

Then feel the arterial pulse and revise its character: slow and prolonged.

4. **Tricuspid regurgitation:** most easily suspected by noting:

- The distended neck veins with marked systolic expansion (v wave).
- Expansile liver (bimanual palpation).
- *Confirm* by listening over tricuspid area for a pansystolic murmur which is increased by inspiration.

Summary

Valve lesion	Most important cause	Most important symptom	Most important sign	Chest X-ray	ECG
MS	Rheumatic	Pulmonary congestion esp. dyspnea	• Accentuated S1. • Mid-diastolic rumbling M over apex. • Pulmonary HTN	• Mitralization. • Pulmonary congestion.	• P-mitrale. • P-pulmonale. • AF.
MR	Rheumatic	Palpitation	• Pan systolic M & thrill over the apex propagated to axilla	• LAE. • LVE.	• LAE. • LVE.
AS	Rheumatic Calcified Congenital	Low CO esp. Angina & syncope	• Heaving apex. • Harsh ejection systolic M & thrill "over the second right space → carotid"	• LVE. • Small aortic knuckle or • Post-stenotic dilatation.	• LVE.
AR	Rheumatic Syphilitic	Throbbing palpitation	• Peripheral signs. • Hyper dynamic apex. • Early diastolic soft blowing M "over the third left space"	• Boot shaped heart.	• LVE.
TS	Rheumatic	Systemic congestion	• Giant a-wave. • Mid-diastolic rumbling M over tricuspid "↑ with inspiration"	• RAE.	• RAE.
TR	Functional	Systemic congestion	• Systolic expansion in neck vein. • Pansystolic M over tricuspid "↑ with inspiration"	• RAE. • RVE.	• RAE. • RVE.

Murmurs

There are two types of heart murmurs:

- Innocent murmurs.
- Abnormal murmurs.

Innocent heart murmurs A person with an innocent murmur has a normal heart.

- This type of heart murmur is common in newborns and children.
- An innocent murmur can occur when blood flows more rapidly through the heart:
 - Physical activity or exercise.
 - Pregnancy.
 - Fever.
 - Changes in your heart's structure, such as changes from heart surgery
 - anemia
 - hyperthyroidism

Innocent heart murmurs may disappear over time, or they may last your entire life without ever causing further health problems.

Abnormal heart murmurs

An abnormal heart murmur is more serious.

- In children, abnormal murmurs are usually caused by congenital heart disease.
- In adults, abnormal murmurs are most often due to acquired heart valve problems.

DD of Diastolic Murmurs

- I. **Aortic regurgitation**
- II. **Pulmonary valve regurgitation (Functional)**
- III. **Mitral rumble**
 - A. Obstruction to flow
 1. Mitral stenosis (rheumatic, congenital)
 2. Left atrial myxoma
 - B. Increased flow
 1. Mitral regurgitation
 2. Ventricular septal defect
 3. Patent ductus arteriosus
- IV. **Tricuspid rumble**
 - A. Obstruction to flow
 1. Tricuspid stenosis (rheumatic, Ebstein's anomaly, carcinoid)
 2. Right atrial myxoma
 - B. Increased flow
 1. Atrial septal defect
 2. Tricuspid regurgitation

DD of Systolic Murmurs**I. Ejection murmurs****A. Functional**

1. Flow murmur from the root of the pulmonary artery
2. Murmur associated with high COP
3. Flow murmurs associated AR - PR

B. Organic

1. Valvular AS
2. Aortic sclerosis
3. Subvalvular AS (web or tunnel)
4. Supravalvular AS
5. Hypertrophic obstructive cardiomyopathy
6. Pulmonary valvular stenosis
7. Pulmonary infundibular stenosis
8. ASD
9. Tetralogy of Fallot

II. Regurgitant murmurs

1. MR
 - a. Rheumatic
 - b. Papillary muscle dysfunction
 - c. Mitral valve prolapse
 - d. Acute
2. TR (**Functional - organic**)
3. VSD

III. Extracardiac sounds simulating systolic heart murmurs

- A. Subclavian (supraclavicular/brachiocephalic) murmur
- B. Internal mammary soufflé
- C. Carotid artery bruits
- D. Coarctation of the aorta**
- E. Murmurs emanating from a dilated aortic or pulmonary artery root
- F. **Patent ductus arteriosus** with pulmonary hypertension

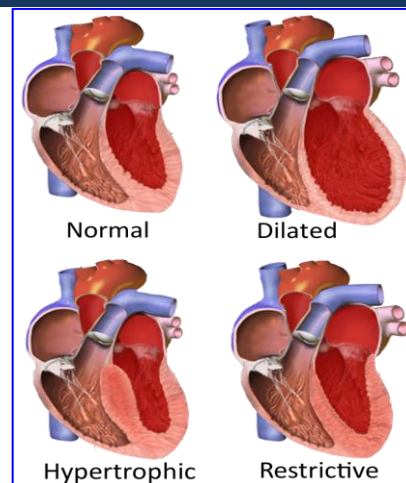
Cardiomyopathy

Definition

group of disorders characterized by primary involvement of the myocardium.

Types

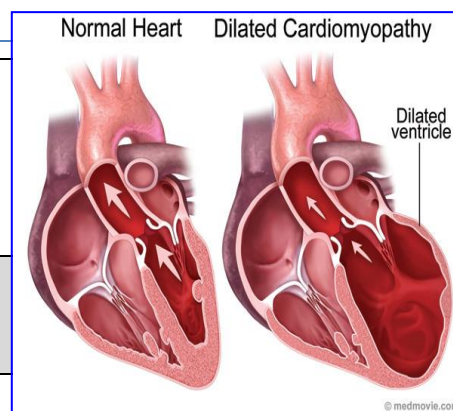
1. Dilated (congestive) cardiomyopathy.
2. Hypertrophic cardiomyopathy (HOCM, IHSS).
3. Restrictive (non-dilated, non-hypertrophic) cardiomyopathy.



Dilated cardiomyopathy

Aetiology

4I	<u>I</u> nfectious: Viral especially coxsackie B virus & Chaga's disease.
	<u>I</u> mmunological e.g. SLE.
	<u>I</u> diopathic (may be Familial).
	<u>I</u> schemic (diffuse myocardial ischaemia).
T	<u>T</u> oxic: Alcohol – Anthracyclines.
E	<u>E</u> ndocrinal: Myxoedema – DM – Acromegaly
N	<u>N</u> eurologic: Duchienne's My, Myotonia & Friedreich's A



Haemodynamics: (LV or both)

- **Systolic Dysfunction** - Decreased Pump.
- **Dilatation.**

Clinical picture

Symptoms	- Low CO. - Pulmonary congestion. - Systemic congestion.	
General	- Low CO. - S. congestion.	
Local	Precordial	- Biventricular enlargement. - Weak apex.
	Auscultation	- Weak heart sounds. - - Murmurs of functional mitral & tricuspid incompetence

Complications

- Arrhythmias: e.g. AF & ventricular tachycardia.
- Embolism: systemic & pulmonary.
- Sudden death.

Investigations

X- Ray	Cardiac enlargement involving all chambers. Pulmonary congestion.
ECG	- Low voltage.- Depressed ST segment.- Flat or inverted T wave.
Echo & Doppler	Dilated ventricles with no hypertrophy - Decreased contractility. Mitral & tricuspid incompetence.

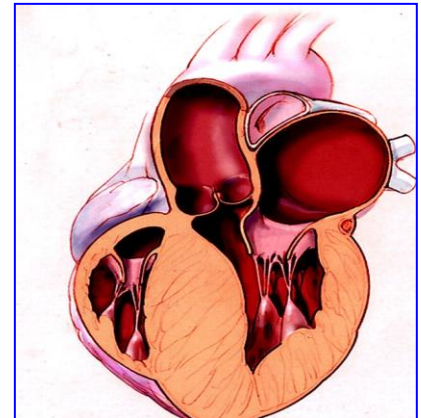
Treatment

- Treatment of HF (may be refractory).
- Treatment of complications.
- Treatment of the cause if possible.

HYPERTROPHIC CARDIOMYOPATHY

Causes:

- Familial: autosomal dominant.
- Idiopathic. (IHSS)
- Pheochromocytoma.
- Young athletes:- may cause sudden death



Hemodynamics: (LV)

- **Diastolic Dysfunction** (i.e. compliance) ---- P. congestion & decreased pump.
- Obstruction of outflow (**low CO**) -
- **Bernhiem effect**:- The septum may encroach on RV cavity ---- mild impairment of filling & giant A wave.

Clinical picture

Symptoms	-Pulmonary congestion. - Low CO.	
General	- Signs of low CO - Pulse: jerky or bisferiens pulse - Giant A wave .	
Local	Precordial	- LV enlargement - Double apex
	Auscultation	- Murmur of MR may be present. - Obstruction of outflow murmur (dynamic)**.

****Midsystolic murmur heard along left sternal border increases with standing & decreases with squatting . (Dynamic). + No Ejection click.**

Complications

- Arrhythmias, especially (AF).
- Sudden death.

Investigations:

X- Ray	- Left ventricular hypertrophy - Pulmonary congestion.
ECG	- Left ventricular enlargement. - Q waves in left chest leads (V5 & V6).
Echo & Doppler	- Hypertrophy of the left ventricle with the (septal wall thickness). - Usually exceeding free wall thickness (diagnostic).

Differential diagnosis

Aortic stenosis.

Treatment

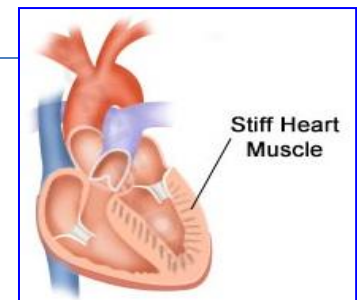
- Treatment of heart failure with avoidance of positive inotropic.
- B-blockers (e.g. Propranolol) or Calcium channel blockers (e.g. verapamil).
- Surgical treatment: resection of septum.

RESTRICTIVE CARDIOMYOPATHY

The wall of the heart has been infiltrated by abnormal tissue.

Causes

- Amyloidosis.
- Sarcoidosis.
- Idiopathic.
- Hypereosinophilic syndrome.
- Haemochromatosis.
- Scleroderma.
- Endomyocardial fibrosis.

**Haemodynamics (RV)**

- Decreased **diastolic compliance** ----- S. congestion.
- No hypertrophy or dilatation (later).

Clinical picture

Symptoms	-S congestion. - Low CO (Late).	
General	- Signs of S. congestion* - low CO (Late)	
Local	Precordial	- RV enlargement (late).
	Auscultation	- Murmur of TR & MR (Papillary Ms fibrosis)

*Congested pulsating neck veins with inspiratory filling

Deep X & Y descents (As, constrictive pericarditis).

Investigations

X- Ray	- RV ++ (late)
ECG	- Low voltage
Echo & Doppler	Decreased ventricular cavity

Differential diagnosis

	Restrictive cardiomyopathy	Constrictive pericarditis
Cardiomegaly	Late	Absent
Calcification	Absent	May be present
CT & MRI		Diagnostic
Endomyocardial biopsy	Diagnostic	
Exploratory thoracotomy	Diagnostic	Diagnostic

Treatment

- Treatment of heart failure which is usually refractory to therapy.
- Treatment of the cause if possible.

Comparison

	Dilated	Hypertrophic	Restrictive
Causes	4 I + TEN	Familial – idiopathic. Pheochromocytoma	Infiltration
Haemodynamics	LV or Both Systolic Dysfunction	LV Diastolic Dysfunction Obstruction of outflow Bernheim	RV Diastolic Dysfunction
Symptoms	Low CO. P. congestion. S. congestion.	P. congestion. Low CO	S. congestion. ± Low CO
General signs	Low CO. S. congestion.	Jerky or bisferiens pulse. Low CO. Giant A wave.	S. congestion. Low CO.
Precordial	LV & RV ++ (weak apex).	LV hypertrophy	RV enlargement.
Auscultation	Weak heart sounds. Functional MR & TR.	Midsystolic dynamic. Murmur of MR.	Murmur of TR & MR
Complications	Arrhythmias. Sudden death or embolization.	Arrhythmias. Sudden death	
D.D.		Aortic stenosis	Constrictive pericarditis
Chest X-ray	All chamber +++ P. congestion.	LV hypertrophy. P. congestion.	RV enlargement.
ECG	Low voltage. Depressed ST Flat or inverted T	LV enlargement. Q waves (V5 & V6).	Low voltage.
Echo	All chambers dilated. Decreased contractility. MR & TR.	Hypertrophy of LV . Septal wall thickness.	Decreased ventricular cavity.

Dysarrhythmias

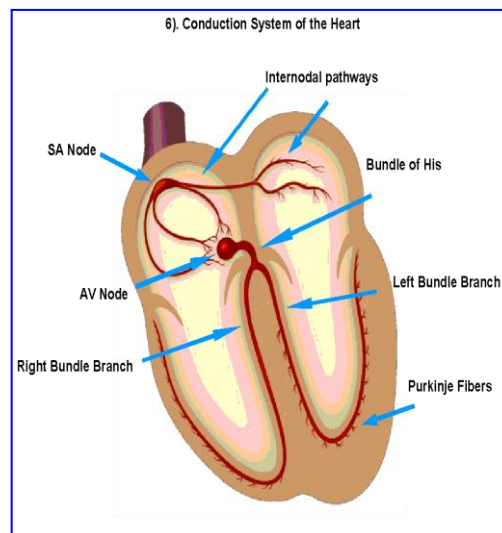
Definition

Any disturbance of Rate, Rhythm or Atrio-ventricular relation.

Physiological Aspects

Normally, the heart rate is 60 - 100 beats/minute - Regular.

- **The SAN (Sino-atrial node):**
 - ✓ It is the normal pacemaker.
 - ✓ At the junction of the SVC and the right atrium.
 - ✓ Sympathetic accelerates, while vagus slows the SAN.
 - ✓ Impulse transmission from the S.A. node through the 3 internuclear bundles.
- **A-Vnode (Atrioventricular node):**
 - ✓ On the atrial side of the annulus fibrosus.
 - ✓ Sympathetic accelerates while vagus slows the conduction in A-VN
 - ✓ Represented by the P-R interval.
 - ✓ No retrograde conduction.
 - ✓ The ventricles are supplied by Sympathetic fibers but not the vagus (vagus escape phenomenon).
- **The right bundle branch** continues down the right ventricular endocardial surface to reach the anterior and apical muscle of the right ventricle.
- **The left bundle branch** crosses the summit of the muscular interventricular septum to emerge on the left side of the heart. The left bundle divides into a left posterior fascicle and a left anterior fascicle.



Classification

A. Aetiological classifications

1. Abnormal automaticity

a. S.A.N.	• Sinus tachycardia. • Sinus bradycardia. • Sinus dysarrhythmia. - Sinus arrest.
b. A.V.N.	• Junctional (nodal) premature beats. • Junctional (nodal) rhythm. • Paroxysmal junctional (nodal) tachycardia.
c. Atrial	• Atrial premature beats. • Paroxysmal atrial tachycardia. • Atrial flutter. • Atrial fibrillation. (AF)
d. Ventricular	• Ventricular premature beats. • Ventricular tachycardia (VT). • Ventricular fibrillation (VF)

2. Abnormal conductivity

A- S.A. block.	
B- A.V. block:	• 1st grade: P-R interval > 0.22 sec. • 2nd grade: Mobitz type one (Wenckenbach phenomenon) & Mobitz type two. • 3rd grade: complete heart block.
C- Bundle block:	• Right bundle branch block (RBBB). • Left bundle branch block (LBBB). • Left anterior hemiblock (LAHB). • Left posterior hemiblock (LPHB).

B. Rhythmic classifications

	Regular	Irregular
Tachy	• Sinus tachycardia. • Paroxysmal supraventricular tachycardia. • Atrial flutter. • Ventricular tachycardia.	• AF. • Sinus tachycardia with premature beats. • Paroxysmal supraventricular tachycardia with variable heart block. • Atrial flutter with variable heart block.
Brady	• Sinus bradycardia. • 1st heart block. • Junctional (nodal) rhythm. • Partial fixed heart block (2:1, 3:1, 4:1, etc.). • Complete heart block.	• Slow partial variable heart block. • Sinus bradycardia with premature beats. • Sinus bradycardia with respiratory sinus dysarrhythmia.

In any cases of Dysrrhythmia

Definition	- Rhythm Rate Origin of beat.....
	± Special features.....

Aetiology

A- General causes:	1. Simple causes (stress, sadness, smoking , coffee and tea) 2. Thyrotoxicosis. 3. Drugs e.g. digitalis, sympathomimetics. 4. Electrolyte disturbances: (eg. Hyperkalaemia).
B- Cardiac causes:	1. Ischaemic heart disease especially myocardial infarction. 2. Myocarditis & cardiomyopathies. 3. Rheumatic & congenital heart diseases.
C- Specific causes:	1. <u>Heart block:</u> • Drugs e.g. digitalis & B-blockers. • Fibrosis & calcification of the AV node & bundle. 2. <u>AF:</u> • <u>MS</u> • Constrictive pericarditis. • Chest diseases e.g. pulmonary embolism, COPD. • Pre-excitation syndromes e.g. WPW syndrome. • Lone AF.

N.B. Sinus dysrrhythmia (3p:- Physiological - Pathological - Pharmacological)

Diagnosis

A- Clinical Picture :

1. Symptoms:

Tachy	Brady
• May be asymptomatic. • Palpitation: onset.....offset.....duration..... and number of recurrence • Symptoms of low CO. • Precipitation of HF in susceptible patients.	
• Thromboembolism especially in AF.	
• Angina • Ventricular arrhythmia may result in sudden death.	• 2nd & 3rd grades heart block may result in Syncope or Sudden death.

2. Signs:

Arterial Pulse (4Rs):

1. Rhythm regular ----- **except** (AF-Extrasystole)
2. Rate.....
3. Response to -----
 - a- **Carotid** stimulation:- slow down HR in all tachy **except Ventricular ??**
 - b- **Excises**:- for brady group

4. Respiratory sinus rhythm: It is the acceleration of the heart rate during inspiration (due to increased venous return) only **positive in sinus rhythm.**

Neck veins (4)

1. Rhythm
2. Rate
3. Waves (A & V waves).....
4. A & V relationship

Heart sound (S1): ($S1 \propto HR$).....

Tachycardia	Accentuated
Bradycardia	Muffled
A-V dissociation	Cannon (occasional)
Junctional	Cannon (regular)
irregular	Variable

Cannon waves & Cannon sounds:

Simultaneous contraction of atria & ventricles results in marked systolic expansion of neck vein (Cannon waves) & very loud first heart sound (cannon sounds).

B- Investigations

1. **ECG:** (Rest ECG or Holter monitoring) Comment on **(4)**:

- a. P wave: rhythm, rate & morphology.....
- b. QRS complex: rhythm, rate & morphology.....
- c. P-R interval
- d. P and QRs relationship.....

P wave	Single P	Sinus arrhythmias .
	Bizarre, inverted or absent	Atrial arrhythmias .
	Multiple	Atrial flutter.
	Absent or fibrillation	Atrial fibrillation.
P-R interval	Short	Tachycardia (mainly WPW syndrome)
	Prolonged	HB (mainly 1st degree)
QRS complex	Bizarre	V. tachy - 3rd HB
	Wide	WPW

2. **Eelectrophysiological studies:**

3. **Diagnosis of the cause of dysrrhythmia:**

Cardiac investigations: e.g. Chest X-ray, ECG, echo & cardiac Catheterization.

Laboratory investigations: e.g. serum K & thyroid function tests.

Treatment

According to the type of dysrrhythmia.

SINUS Tachycardia

Definition:

It a condition in which the SAN discharges at a high rate ($> 100 / \text{min}$) & regular rhythm.

Aetiology (3Ps):

1. **P**hysiological: - Exercise, emotional stress, pregnancy & infancy.
2. **P**athological: Fever, hyperdynamic circulation, heart failure, shock & myocarditis.
3. **P**harmacological: e.g. adrenaline, atropine, thyroxine, nifedipine.

Clinical picture

Symptoms

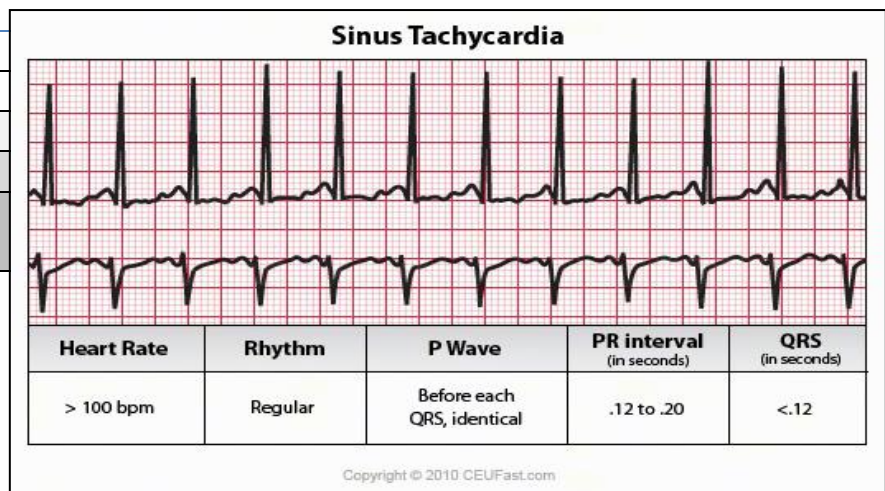
1. May be asymptomatic.
2. Palpitation: rapid regular palpitation of gradual onset.
3. Symptoms of low CO may occur in severe cases.
4. May precipitate angina & heart failure in susceptible patients.

Signs

Pulse	Rhythm	Regular
	Rate	100-180
	Response to	Carotid:- Gradual slowing then gradual return to original HR
	Res. sinus rhythm	Positive
Neck V	Rhythm	Regular
	Rate	100-180
	Wave	Normal
Auscultation	S1	Accentuated

Investigation

ECG	Rhythm	Regular
	Rate	100-180
	P-R	Short
	Waves	Each P followed by a QRS



Treatment

1. Treatment of the cause.
2. Sedation may be needed.
3. B-blockers may be given if there is no contraindication e.g. propranolol.

Paroxysmal supra ventricular tachycardia(PSVT)

Definition

- It is a dysrhythmia originating from the atrium (atrial tachycardia) or the AVN (junctional tachycardia).
- It is characterized by being regular, rapid, of sudden onset, sudden offset, short duration and tendency for recurrence (Paroxysmal).

Aetiology (as usual + WPW)

Cardiac causes

- Ischaemic heart disease especially myocardial infarction.
- Myocarditis & cardiomyopathies.
- Rheumatic & congenital heart diseases.

General causes

- Simple causes (stress, sadness, smoking, coffee and tea).
- Thyrotoxicosis.
- Drugs e.g. digitalis, sympathomimetics.
- Electrolyte disturbances: (e.g Hyperkalaemia)

Specific causes:

- Pre-excitation syndromes e.g. **WPW** syndrome.

May Occur in apparently normal individuals.

Clinical Picture

Symptoms

- Never to be asymptomatic. **Paroxysmal**
- Palpitation: rapid regular palpitation of sudden onset, sudden offset, short duration and tendency for recurrence.
- Symptoms of low CO may occur in severe cases.
- May precipitate angina & heart failure in susceptible patients.

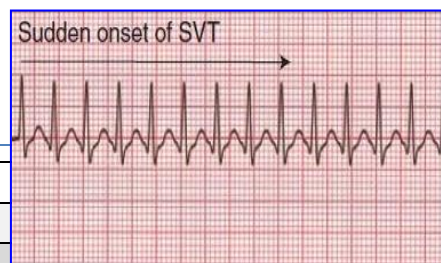
Signs

Pulse

Pulse	Rhythm	Regular
	Rate	150-250
	Response to	Carotid :- reversion to initial HR may occur in some patients
	Res. sinus rhythm	negative
Neck V	Rhythm	Regular - 150-250
	Rate	* Atrial tachycardia : normal waves with the same rate of pulse
	Wave	* Junctional tachycardia : regular <u>cannon</u> with the same rate of pulse
Auscultation	S1	* Atrial tachycardia : accentuated S ₁ .
		* Junctional tachycardia : regular cannon, sounds.

Investigations

ECG	Rhythm	Regular
	Rate	150 - 250
	Waves	QRS :- normal morphology P wave :- - Deformed in <u>Atrial tachycardia</u> - Inverted or absent in <u>Junctional tachycardia</u>



	Atrial tachycardia	Junctional tachycardia
Neck vein		
Rhythm.	Regular	
Rate	With the same rate of pulse (150-250/min)	
Waves	Normal	Cannon
Auscultation		
S1	Accentuated	Cannon
ECG		
Rhythm:	Regular	
Rate	With the same rate of pulse (150-250/min)	
QRS	Normal morphology	
P Wave	Deformed	Inverted or absent

Treatment**A. During the attacks:**

1. Vagal stimulation: using carotid sinus massage (or less preferably ocular compression or induction of vomiting).
2. If there is no response: Verapamil 5-20mg IV or Digitalis IV or Propranolol IV.
3. If there is no response and the patient is haemodynamically unstable (e.g. presence of hypotension or heart failure): DC is indicated.

B. In between the attacks:

1. Treatment of the underlying causes.
2. Maintenance therapy with antiarrhythmic drugs e.g. verapamil, digitalis, Propranolol or quinidine orally.
3. In (resistant case) of pre-excitation syndromes: division of the accessory pathway is indicated.

Atrial flutter

Definition

It is a condition in which the **atria** discharge at a **regular** rapid rate (**250 - 350/min**)

- Due to the physiological block at the AVN only 1/2, 1/3, 1/4, etc of the atrial impulses are transmitted to the ventricles (**A rate > V rate**)

Aetiology (As, usual + WPW)

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea)
2. Thyrotoxicosis.
3. Drugs e.g. digitals, sympathomimetics.
4. Electrolyte disturbances: (eg, Hyperkalaemia).

Specific causes:

- Pre-excitation syndromes e.g. **WPW** syndrome.

Clinical Picture

Symptoms:

1. Palpitation: rapid regular palpitation of sudden onset, sudden offset, short duration and tendency for recurrence.
2. Symptoms of low CO may occur in severe cases.
3. May precipitate angina & heart failure in susceptible patients

N.B. The condition may change to sinus spontaneously, or progress to AF.

Signs

Pulse	Rhythm	Regular
	Rate	120-180 depending on the degree of A-V conduction
	Response to	Carotid:- HR decreases in a mathematical fashion (e.g. 150-100- 75) On release:- the rate returns to its initial HR by the same fashion
	Res. sinus rhythm	Negative
Neck V	Rhythm	Regular
	Rate	120-180
	Wave	Multiple A waves before each V according to A/V ratio.
Auscultation	S1	Accentuated

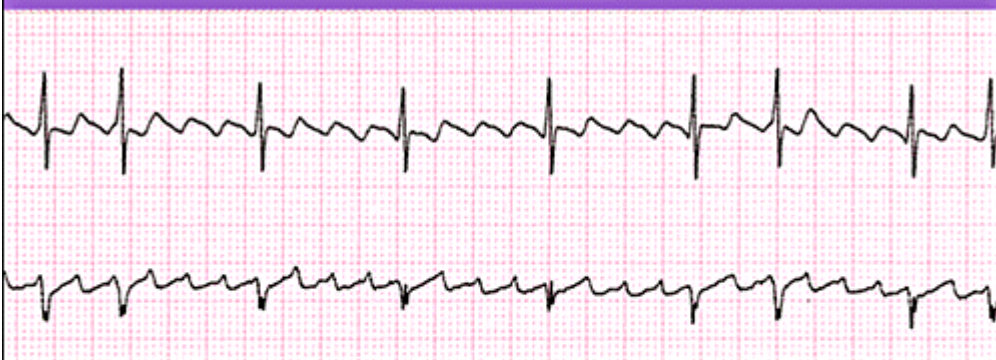
Investigations

ECG	Rhythm	Regular
	Rate	120-180
	Waves	Multiple P waves before each QRS (Saw-tooth appearance)



Treatment

1. **In haemodynamically unstable patient** (e.g. presence of hypotension or heart failure): Cardioversion by Overdrive pacing is indicated.
2. **Haemodynamically stable patient:** Verapamil 5 - 20 mg IV or Propranolol IV.
3. **Digitalis:** could be given to slow the ventricular rate by decreasing conduction in the AVN. After stoppage of digitalis atrial flutter is converted to AF or sinus rhythm may be restored.
4. **For prevention of recurrence:** class I antiarrhythmic (e.g. quinidine) alone or with digitalis is usually given.

Atrial Flutter				
				
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

Ventricular tachycardia (Paroxysmal)

Definition

It is a dysrhythmia originating from the ventricles & characterized by being **regular** rapid, of sudden onset, sudden offset, short duration and tendency for recurrence (**Paroxysmal**)

- The ventricles will be controlled by the (ectopic focus) while the atria controlled by the SAN (A-V dissociation).

Aetiology

Cardiac causes:

- Ischaemic heart disease especially myocardial infarction.
- Myocarditis & cardiomyopathies.
- Rheumatic & congenital heart diseases.

General causes:

- Simple causes (stress, sadness, smoking, coffee and tea).
- Thyrotoxicosis.
- Drugs e.g. digitalis, sympathomimetics.
- Electrolyte disturbances: (e.g. Hyperkalaemia).

Clinical Picture

Symptoms

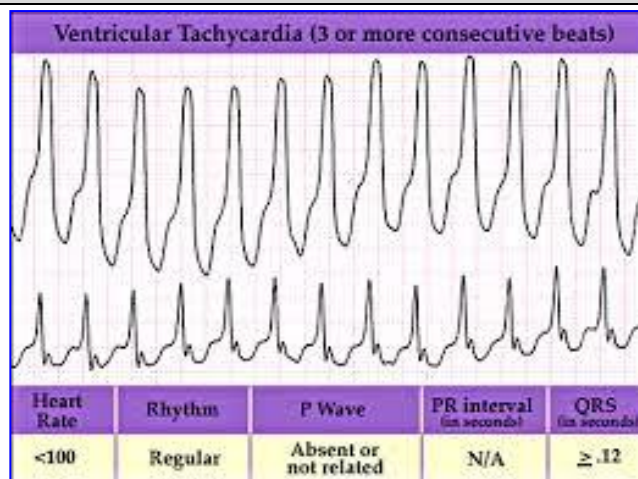
- Never to be asymptomatic.
- Palpitation: rapid regular palpitation of sudden onset, sudden offset, short duration and tendency for recurrence.
- Symptoms of low CO may occur in severe cases.
- May precipitate angina & heart failure in susceptible patients.
- Sudden death** (if converted to ventricular fibrillation).

Signs

Pulse	Rhythm	Regular
	Rate	100 - 220
	Response to Carotid:-	no effect (Vagal escape phenomenon)
	Res. sinus rhythm	Negative
Neck V	Rhythm	Regular
	Wave	Occasional Cannon waves Rate of A wave: 60-100/min - No relation between A wave and V wave.
Auscultation	S1	Variable with occasional cannon sound

Investigations

ECG	Rhythm	Regular
	Rate	100-220
	Waves	QRS complex: of rapid regular rate and <u>deformed (bizarre)</u> no relationship between P & QRS complex



Treatment

A. During the attack

1. If the patient is hemodynamically stable:

- Amiodarone or Procainamide or amiodarone: IV.
- Lidocaine: initial bolus of 2 mg / kg IV, followed by infusion of 1 – 4 mg / min.
(it was the drug of choice for a long time in all cases but now, it is only considered in peri-infarction V. tach - adenosin is CI)
- Bretylium: IV (in resistant cases).

2. If the patient is hemodynamically unstable:

DC cardioversion immediately, followed by Pharmacological ttt.

B. Prevention of a future attack:

1. Maintenance therapy:

Drugs: (oral), class one or class three anti-arrhythmic drugs.

2. Intervention: Ablation of focus: **Catheter** (radiofrequency energy) or **surgery**.

Implantable Cardioverter Defibrillator (ICD): "Anti- tachycardia pacing".

3. Treatment of the cause.

Rapid regular

	Sinus tachycardia	PSVT		Atrial flutter	PVT
Definition					
Rhythm	Regular				
Rate	> 100 / min	150 – 250	250 – 350	100 – 220	
Origin of beat	SAN	Atrial or nodal	Atrial	Ventricular	
Special features			Reductive conductivity	A-V dissociation	
Aetiology	3Ps	As usual + WBW			As usual
Clinical pictures					
Symptoms	As usual	As usual (never asymptomatic).	As usual	As usual (never asympt).	
Signs					
Arterial pulse (4Rs)					
Rhythm	Regular				
Rate	> 100 / min	150 – 250 / min	250 – 350	10 – 220	
Response to Carotid	Gradual slowing		Mathematical reduction in HR	No effect	
R. S. rhythm	Positive	Negative	Negative	Negative	
Neck vein (4)					
Rhythm	Regular				
Rate	> 100 / min	Atrial	Nodal		
Waves	Normal	Normal	Reg. cannon	A > V	
A & V relationship	Normal	As pulse		No relation	
Heart sounds S ₁	Accentuated	Accent	Reg. cannon	Accentuated	
ECG					
P wave	Normal	Deformed	Inverted	Multiple small (saw tooth)	Normal
QRS	Normal	normal		Normal	Deformed (50%)
P-R interval	Normal	Normal		Normal	Normal
P and QRS relationship	Each P followed by QRS	Each P followed by QRS	P > QRS		No relation

Atrial fibrillation (AF)

Definition

It is a condition in which the atria discharge impulses irregularly usual at a high rate of 400-600/min.

Due to the physiological block in the AVN, the ventricular rate is less than the atrial rate.

Hemodynamic Effects

Atrial fibrillation is associated with the loss of the atrial contribution to ventricular filling. This may result in a decrease in ventricular stroke volume of up to 20%.

Aetiology

Cardiac causes:-

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea).
2. Thyrotoxicosis.
3. Drugs e.g. digitalis, sympathomimetics.
4. Electrolyte disturbances: (eg. Hyperkalaemia).

Specific causes:

1. MS
2. Constrictive pericarditis.
3. Chest diseases e.g. pulmonary embolism, COPD.
4. Pre-excitation syndromes e.g. **WPW** syndrome.
5. Lone AF*.

The term "**lone atrial fibrillation**" describes atrial fibrillation in the absence of demonstrable underlying cardiac disease or a history of hypertension. It may be due to fibrotic areas in the atrium that predisposes patients to arrhythmia.

Clinical Picture

Symptoms:

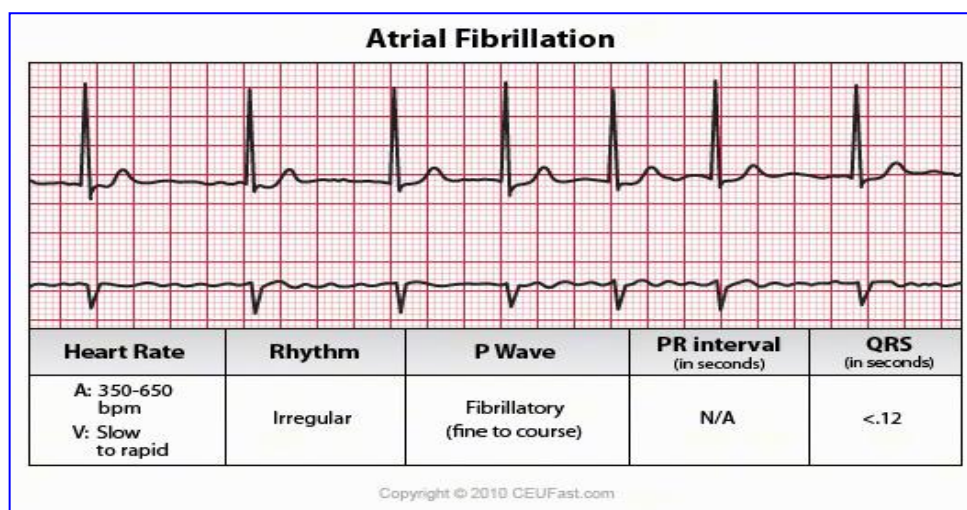
1. Palpitation: rapid irregular, may be paroxysmal or persistent.
2. Symptoms of low CO.
3. Precipitation of angina & heart failure in susceptible patients.
4. May cause atrial thrombosis & embolizations.

Signs

Pulse	Rhythm	Marked irregularity
	Rate	Usually rapid e.g. 100 - 150/min. Slow AF: heart block - patients receiving digitalis or propranolol - lone AF*
	Response to	Carotid: slowing of pulse due to decreased AV conduction. Exercise: increased irregularity due to increased AV conduction
	Res. sinus rhythm	Negative
Pulse deficit: more than 10 beats/min		
Neck V	Rhythm	Marked irregularity
	Rate	Usually rapid except
	Wave	absent A wave with prominent V (Systolic expansion)
Auscultation	S1	Marked irregularity & variability

Investigations

ECG	Rhythm	markedly irregular rhythm
	Rate	Usually rapid except
	Waves	Absent P waves which are replaced by irregular vibrations (fibrillation waves) QRS complexes are of normal shape & markedly irregular rhythm

**Types of AF**

1. **Newly Diagnosed Atrial Fibrillation**
2. **Recurrent Paroxysmal Atrial Fibrillation.**
3. **Persistent Atrial Fibrillation:**
Once an episode of atrial fibrillation has lasted more than **seven days**, spontaneous conversion is rare and the condition can be defined as persistent.
4. **Drug-Refractory Atrial Fibrillation:**

Treatment of AF

either (Rate control or Rhythm control)

A. Treatment of the cause.**B. Cardioversion to sinus****Rhythm control****1. Indicated in the following conditions:**

- Recent AF (less than 12 months duration).
- No history of embolism.
- No marked enlargement of atria.

2. Types:

- Electrical & Chemical by quinidine.

	Electrical	Chemical
<i>should be preceded by</i>	Discontinuation of digitalis	Anticoagulation
<i>Procedure</i>	<u>Electrical cardioversion</u> (of choice)	<u>Quinidine:</u> 1. 1 tablet (0.2 gm) as test for hypersensitivity. 2. (2 tab / 2 h) until sinu-version or 5 doses. 3. IF failed:- larger doses could be used in the second day. 4. In toxicity, quinidine should be stopped. 5. Maintenance dose:- half dose of sinu-version dose.

Rate control**c. Control of ventricular rate-** (Reversion to sinus rhythm is not indicated)

- Long standing AF.
- History of embolism.
- Marked enlargement of atrium.

TTT: Digitalis, verapamil or propranolol are indicated to slow the ventricular rate.

D. Anticoagulation: is indicated in following condition:

- Before reversion to sinus rhythm
- Previous embolizations.
- Valvular diseases as MS.

Premature beats (Extrasystoles)

Definition

One of the irregular dysrhythmias due to ectopic impulses arising from the atria, AVN or ventricles - causing premature beats followed by compensatory pause.

Aetiology

May occur in normal person: e.g. due to anxiety, excessive smoking, coffee & tea.

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea)
2. Thyrotoxicosis
3. Drugs e.g. digitalis, sympathomimetics.
4. Electrolyte disturbances: (eg. Hyperkalaemia).

Clinical Picture

Symptoms

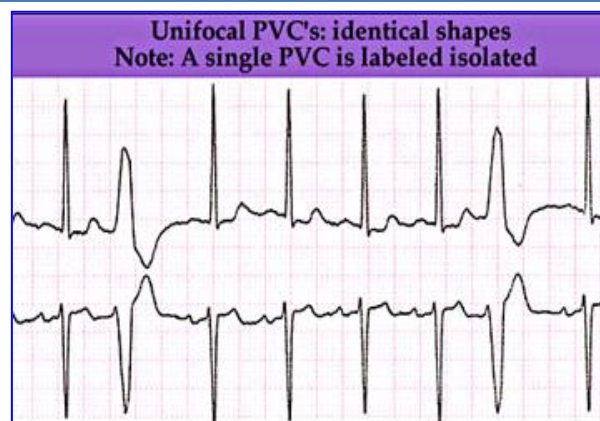
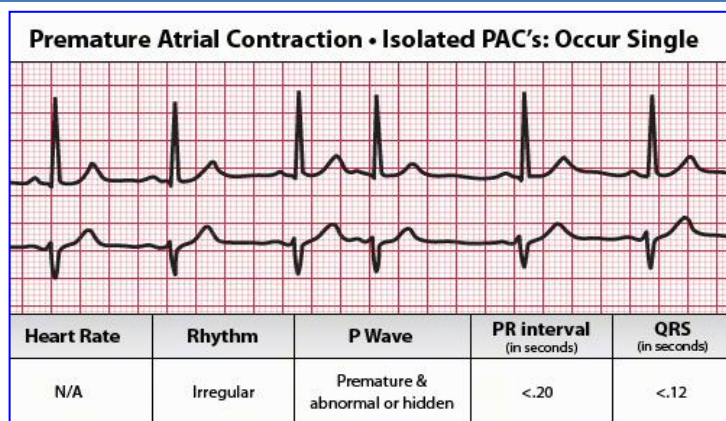
- May be asymptomatic.
- Irregular palpitation. (Described by the patient as an occasional filling of missed or repeated beats).

Signs

Pulse	Rhythm	Mild (occasional) irregularity
	Rate	<u>Sinus</u>
	Response to	Exercise: decrease irregularity due to shortening of diastole
	Res. sinus rhythm	Positive
	Pulse deficit: Less than 10 beats/min	
Neck V	Rhythm	Occasional irregularity
	Rate	Sinus
	Wave	Normal waves
Auscultation	S1	normal sounds with Occasional irregularity

Investigations

ECG	Rhythm	Mild irregular rhythm
	Rate	Sinus
	Waves	Prematurely beat followed by a compensatory pause Supraventricular: inverted or absent P wave followed by normal QRS complex. Ventricular: deformed QRS not preceded by P wave.



Differential diagnosis of irregular tachycardia

	Extrasystole (with S. tachycardia)	AF
Pulse		
Rhythm	Occasional irregularity	Marked irregularity
Rate	According to sinus	Usually rapid e.g. 100 – 150 / min
Carotid massage		± Slowing of pulse
Exercise	Decrease irregularity	Increased irregularity
Pulse deficit	< 10 beats / min	> 10 beats / min
Respiratory sinus rhythm	Positive	Negative
Neck vein		
A wave	Normal with occasional irregularity	Absent
V wave		Systolic expansion
Heart sounds		
	Normal with occasional irregularity	Variability
ECG		
P	Premature beat followed by compensatory pause	Absent
QRS		Marked irregular

Treatment

- Treatment of the underlying cause.
- Sedation may be needed
- Antiarrhythmic drugs are indicated if premature beats were:
 1. Multiple.
 2. Multifocal.
 3. Occurring in runs.
 4. R on T phenomenon.
 5. Causing symptoms.
 6. In association with acute myocardial infarction.

Amiodarone, β blocker, Ca channel blocker or quinidine are used except in myocardial infarction, In which lidocaine is indicated.

Wolf-Parkinson-White (WPW) syndrome (Pre-excitation)

Def:- It is an accessory pathway between atrium & ventricle (bypass the AVN).

C/P:-

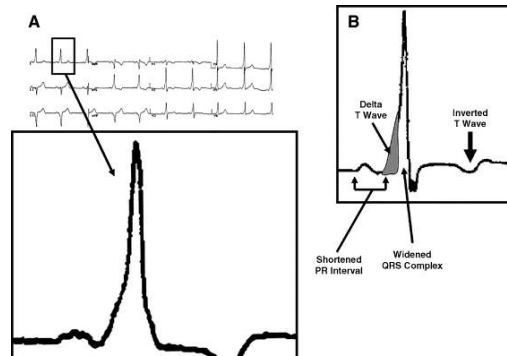
- More in men - associated with MVP, thyrotoxicosis or cardiomyopathy
- Usually asymptomatic
- Or any supraventricular tachycardia:- (S. tachy-PSVT-AF)

ECG:- Short P-R interval - wide QRS complex - delta wave

Treatment :

Medical :- Amiodarone , β blocker (Digitalis is contraindicated).

Surgery:- Ablation.



Sick sinus syndrome (SSS):- rare dysfunction of SA node
may be presented with:-

- Sinus bradycardia
- Sinus arrest (junctional rhythm)
- Atrial fibrillation (slow form)
- Ectopic atrial tachycardia -- prolonged asystolic period.

Sinus bradycardia

Definition

It is a condition in which the SAN discharges at a slow rate $<60/\text{min}$ & regular rhythm.

Aetiology (3Ps)

Physiological: During sleep & in athletes.

Pathological: Myxoedma, obstructive jaundice, post-viral, increased ICT, excessive vagal stimulation & sick sinus syndrome (????).

Pharmacological: B-blockers, digitalis, verapamil or reserpine.

Clinical Picture

Symptoms

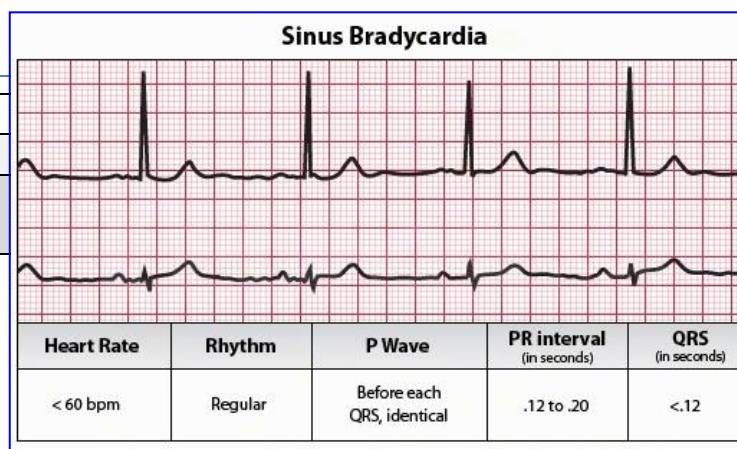
- May be asymptomatic - Palpitation: rare
- Symptoms of low CO may occur in severe cases.
- May precipitate heart failure in susceptible patients.

Signs

Pulse	Rhythm	Regular
	Rate	50-60/min & may be less
	Response to	Exercise: gradual acceleration then gradual returns to the HR
	Res. sinus rhythm	Positive
Neck V	Rhythm	Regular
	Rate	50-60/min & may be less
	Wave	Normal
Auscultation	S1	Normal S1 (may be muffled).

Investigation

ECG	Rhythm	Regular
	Rate	50 - 60/min & may be less
	Waves	Each P followed by a QRS



Treatment

1. Treatment of the underlying causes.
2. Atropine is given in symptomatic cases.
3. Presence of marked symptoms may require pacing.

Junctional (nodal) rhythm

Definition

It is a condition in which the (AVN) becomes the pacemaker of the heart. The impulses spread to activate the atria & ventricles simultaneously.

Aetiology:

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

N.B. It may occur in apparently normal individuals.

Clinical Picture

Symptoms

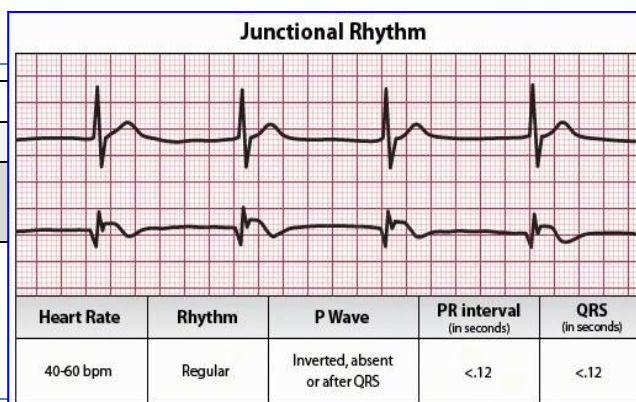
- May be asymptomatic
- Palpitation : rare.
- Symptoms of low CO may occur in severe cases.
- May precipitate heart failure in susceptible patients.

Signs

Pulse	Rhythm	Regular
	Rate	40-50/min & may be less
	Response to Exercise:	reversion to initial HR may occur in some patients
	Res. sinus rhythm	Negative
Neck V	Rhythm	Regular
	Rate	40-50/min & may be less
	Wave	Regular cannon waves with the same rate of pulse.
Auscultation	S1	Regular cannon sound.

Investigations

ECG	Rhythm	Regular
	Rate	40 - 50/min & may be less
	Waves	P wave is inverted or absent QRS normal morphology



Treatment

1. Treatment of the underlying causes.
2. Atropine is given in symptomatic cases.
3. Presence of marked symptoms may require pacing.

Heart block (HB)

A-V Block

- 1st Grade: P-R interval > 0.22 sec.
- 2nd Grade:
 - ✓ Mobitz type one: **Wenckebach** phenomenon.
 - ✓ Mobitz type two.
- 3rd Grade: Complete heart block.

Bundle block

- Right bundle branch block (RBBB).
- Left bundle branch block (LBBB).
- Left anterior hemiblock (LAHB).
- Left posterior hemiblock (LPHB).

ATRIOVENTRICULAR BLOCK

Definition

Delayed conductivity through AV node.

Aetiology

Cardiac causes

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes

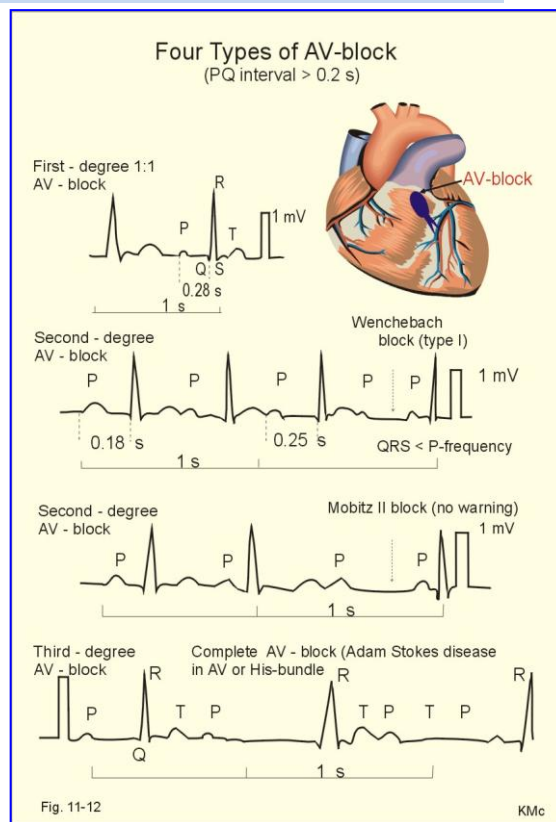
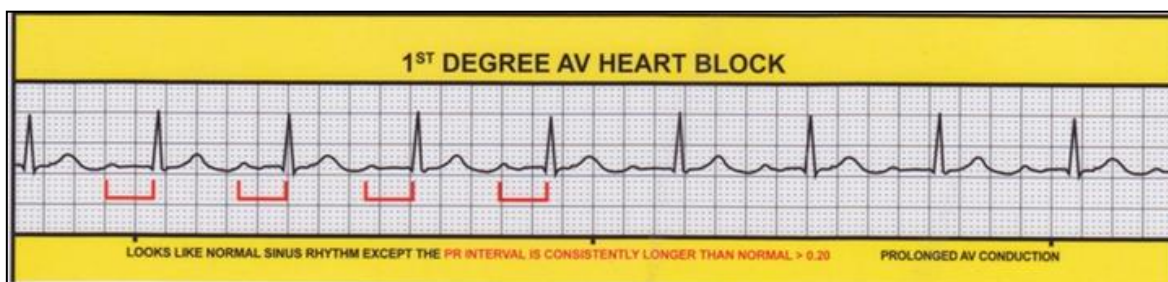
1. Simple causes (stress, sadness, smoking, coffee and tea)
2. Electrolyte disturbances: e.g., Hyperkalaemia)

Specific causes

1. Drugs e.g. B-blockers
2. Fibrosis & calcification of the AV node & bundle (as in calcific AS)

First Grade A-V Block

- It is **prolongation** of P-R interval more than **0.22 sec**.
- Clinically **asymptomatic** but weak S₁ may be detected.
- Treatment: treatment of the underlying cause & **atropine** may be given.



Second Grade A-V Block

There are two types:

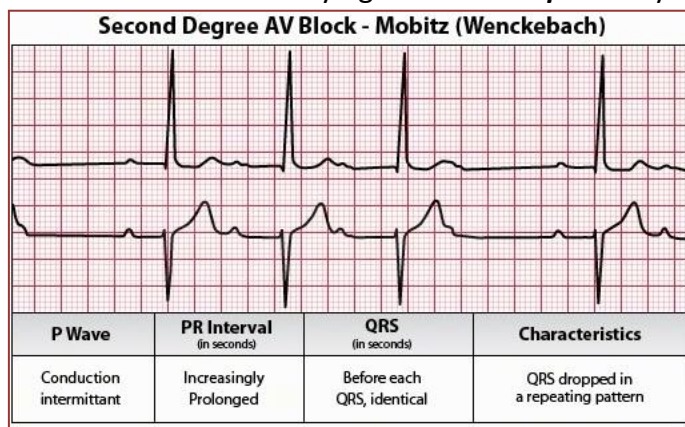
1. Mobitz type 1 (Wenckebach phenomenon):

Definition

- Progressive prolongation of the P-R interval.
- Followed by dropped QRS complex.
- The sequence is repeated.

Clinically:- There is irregular pulse & variable S₁.

Treatment:- Treatment of the underlying cause & **atropine** may be given.



2. Mobitz type 2:

- The AVN transmits one impulse for each 2, 3, 4 or more atrial activities.
- The block may be fixed (e.g. 2:1, 3:1) or variable.

Clinical Picture

Symptoms

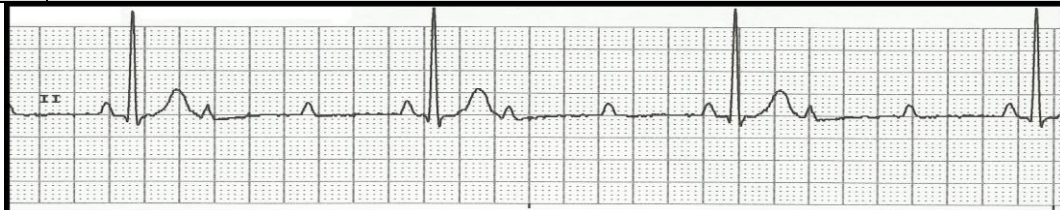
- May be **asymptomatic**. - Palpitation: **rare**.
- Symptoms of low CO may occur in severe cases.
- May precipitate **heart failure** in **susceptible patients**.
- **Adams-Stokes attack** may occur during transmission from partial to complete heart block.

Signs

Pulse	Rhythm	may be regular or irregular (Variable conductivity)
	Rate	40-50/min & may be less
	Response to	Exercise: increase in mathematical fashion then returns to its original level by the same fashion
	Res. sinus rhythm	positive (Hardly noticed)
Neck V	Rhythm	regular or irregular
	Rate	30-50/min & may be less
	Wave	multiple A waves before each V
Auscultation	S1	Normal S1 (may be muffled).

Investigations

ECG	Rhythm	regular or irregular
	Rate	40 - 30/min
	Waves	Multiple Ps (2, 3 or more P waves) between each successive QRS



Treatment

1. Treatment of the underlying causes.
2. Parasympatholytics: Atropin 0.5 - 2.
3. Sympathomimetics: Isoprenaline 0.5-5 ug /min IV.
4. Presence of marked symptoms may require pacing.
5. Treatment of heart failure if present.

Third grade heart block (complete heart block)

Definition

There is complete absence of A-V conduction, so the atria will be controlled by the SAN while the ventricles are controlled by an idioventricular rhythm (A-V dissociation).

Clinical Picture

Symptoms

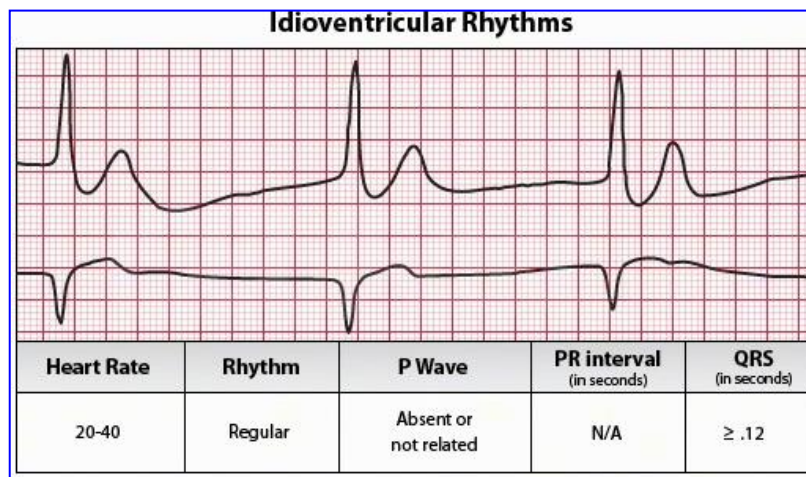
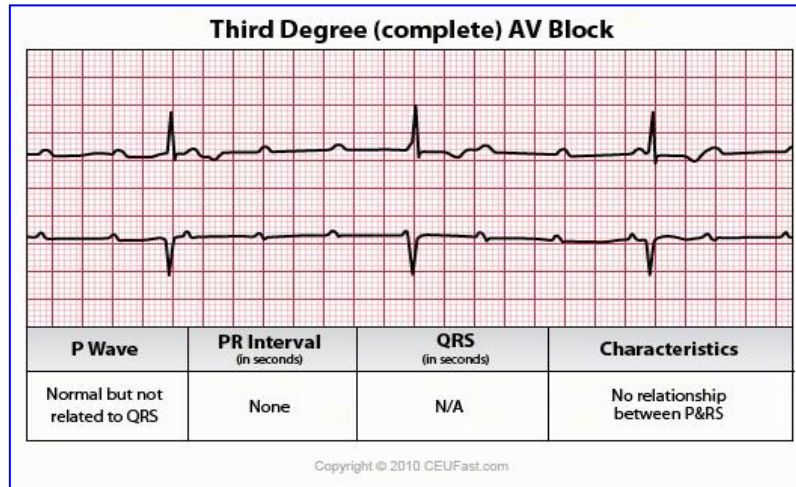
1. May be asymptomatic. - Palpitation: **rare**.
2. Symptoms of **low CO** may occur in severe cases.
3. May precipitate **heart failure** in **susceptible** patients.
4. **Adams-Stokes attack** may occur during transmission from one idioventricular focus to another (During the attack, there is syncope. Absent pulse & low or absent BP and cyanosis. If the attack is prolonged, convulsions, coma & death may occur.)

Signs

Pulse	Rhythm	Regular
	Rate	25-40/min & may be less
	Response to Exercise:	Better to be avoided or no effect
	Res. sinus rhythm	Negative
Neck V	Rhythm	regular
	Wave	with occasional cannon A waves of normal rate (60-100/min)
Auscultation	S1	Normal S1 (may be muffled) with occasional cannon.

Investigations

ECG	Rhythm	Regular & slow
	Rate	25-40/min
	Waves	The QRS complexes are widened, bizarre or may be normal . The P waves are of normal rate (60 - 100/min) & morphology and may be superimposed on QRS (A - V dissociation).



Treatment

1. Treatment of the underlying causes.
2. Parasympatholytics: Atropin 0.5 - 2
3. Sympathomimetics: Isoprenaline 0.5 - 5 ug / min IV.
4. Presence of marked symptoms may require pacing.
5. Treatment of heart failure if present.

Antiarrhythmic agents (Vaughan Williams classifications)

Class	Examples	Clinical uses
I (Na⁺) blocker		
a	- Quinidine - Disopyramide	- Broad spectrum - Procainamide in <u>WPW</u>
I-b	- Lidocaine - Mexiletine	- Ventricular tachycardia (with risk of V. asystole)
I-c	- Encainide - Propafenone	- Broad spectrum (2nd line) - Cl:- post-MI.
II Beta-blockers	- Propranolol - Timolol - Atenolol	- Tachyarrhythmias - decrease <u>MI</u> mortality
III K-blockers <i>Prolonging repolarization</i>	- Amiodarone - Ibutilide - Dronedarone	Broad spectrum WPW (sotalol)
IV Ca-blockers	- Verapamil - Diltiazem	Supraventricular arrhythmias
V <i>Q; Direct nodal inhibition</i>	- Adenosine - Digoxin - Magnesium Sulfate	Supraventricular arrhythmias (mainly in HF) Cl:- V. arrhythmias.

Class one A

	Effect	Indication	Side effects
Quinidine	- Decrease conduction and excitability. - Slow S.A. node. - Vasodilation.	- PSVT. - AF. WPW. V. tachycardia.	- Hypotension & tachycardia. - Depresses vagal tone. Cinchonism (GITD, tinnitus, headache, auditory and visual disturbances and vertigo). - Hypersensitivity.

Class one B

	Effect	Indication	Side effects
Lidocaine	Depression of rapidly depolarizing tissue.	V. tachycardia. - VF - W.P.W.	- C.N.S. effects: - Muscle twitching - Mentality:- psychosis and seizures. - CVS:- suppression

Class two :- B-Blocker (see IHDs and HTN)

Class three

	Effect	Indication	Side effects	Contraindications
Amiodarone has a low incidence of pro-arrhythmic effects	Reduce heart rate.	- Life-threatening ventricular arrhythmias. - supraventricular arrhythmias	- Pulmonary toxicity and fibrosis. - Liver disease. - C.N.S. symptoms. - Hypothyroidism or hyperthyroidism. - Cutaneous photosensitivity and blue-gray discoloration. - Peripheral neuropathy. - Increased levels of LDL.	2nd or 3rd degree heart block. - cardiac shock

Class four *Ca-Blocker (see IHDs and HTN)***Miscellaneous**

	Effect	Indication	Side effects	Contraindications
Adenosine	Decrease AV nodal conduction.	- PSVT. (Used acute)	- Flushing. - Shortness of breath. - Atrioventricular block. - Transient AF. - Headache. - GI upset. - Hypotension. - Paresthesias.	- Heart block. SSS.

Aortic aneurysm

Definition

Enlargement (dilation) of the aorta to greater than 1.5 times normal size.

Causes

1. Atherosclerosis (the most common cause).
2. Congenital.
3. Mycotic.
4. Syphilitic.
5. Traumatic.

Site:

- Ascending aorta.
- Arch of aorta.
- Descending thoracic or abdominal aorta.

Clinical Picture:

A. Aneurysm of the ascending aorta:

It gives rise to marked signs & few symptoms:

Symptoms

1. Chest pain may occur.
2. Huge aneurysm:- mediastinal syndrome.
3. Thrombo-embolism.
4. Rupture:- fatal haemorrhage.

Signs:

Precordial examination.

- Over second right intercostals space
 1. Systolic expansile pulsations.
 2. Palpable second sound & systolic thrill.
 3. Dullness over the second right intercostals space.
 4. Left ventricular enlargement may be detected (associated AR).

Auscultation:

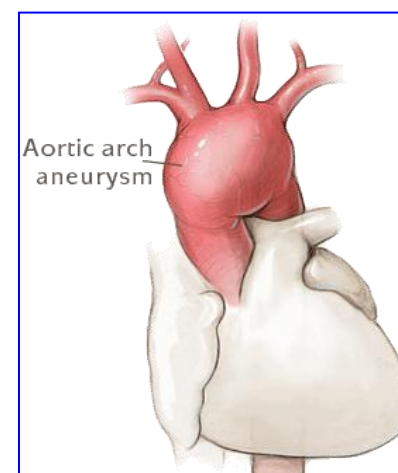
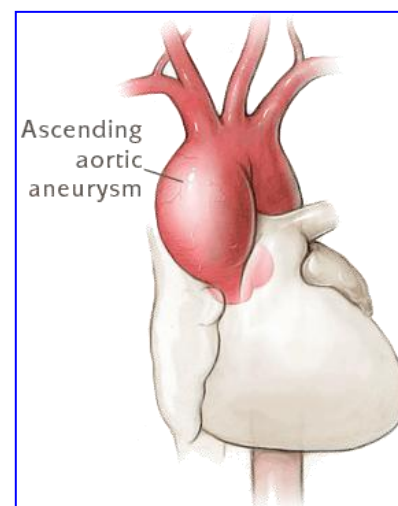
- Over the aortic area reveals:
 - ✓ S2 Loud ringing.
 - ✓ Early diastolic murmur (aortic incompetence).
 - ✓ Ejection systolic murmur (relative aortic stenosis).

B. Aneurysm of the arch of the aorta:

Marked symptoms & few signs:

Symptoms:

1. Chest pain may occur.
2. Mediastinal syndrome.
3. Thrombo-embolism.
4. Rupture of the aneurysm leading to fatal hemorrhage (through the trachea, esophagus, pericardium, pleura, mediastinum or chest wall).



Signs:

1. Systolic pulsations in the suprasternal notch, left infraclavicular region & second left intercostals space.
2. **Tracheal tug:** downward pull on the cricoids cartilage synchronous with the heart beats due to pressure on the bronchi.

Investigations**1. Chest X-ray & Screen:**

- Dilatation of the aorta (fusiform or saccular).
- Calcification in the wall may be seen.
- Pulsations of the aneurysm under fluoroscopy.

2. Echocardiography:**3. CT scan & MRI****4. Aortography:** diagnostic**5. Investigations for the cause:** e.g. serological test for syphilis.**Treatment**

1. Avoidance of **exertion**.
2. Treatment of the **cause** if possible.
3. **B-blockers** may decrease the rate of enlargement of the aneurysm.
4. **Surgical** treatment: resection of the aneurysm & replacement with a prosthetic graft is indicated in some patients.

Aortic dissection

Definition

A serious condition in which the inner layer of the aorta tears. Blood surges through the tear, causing the inner and middle layers of the aorta to separate (dissect). If the blood-filled channel ruptures through the outside aortic wall, aortic dissection is often fatal.

Causes

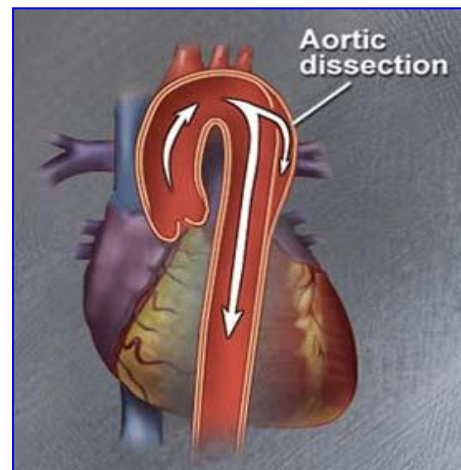
1. Hypertension.
2. Cystic medial necrosis & Marfan's syndrome.
3. Pregnancy.
4. Aortic stenosis & coarctation.
5. Trauma.

Classification

- Type one: affecting ascending, arch & descending aorta.
- Type two: affecting ascending aorta only.
- Type three: affecting descending aorta.

Clinical picture

1. Acute severe chest pain which may radiate to interscapular area.
2. Pain may in the neck & jaw (if the aortic arch is involved).
3. Nausea, vomiting & excessive sweating.
4. Acute aortic regurgitation.
5. Rupture in pericardium leading to haemopericardium.
6. Involvement of branches of aorta:
 - a. Carotid arteries: neurological manifestations & unequal carotid pulsations.
 - b. Subclavian arteries: unequal pulse & **low BP** in upper limbs.
 - c. Coronary arteries: ischaemia & infarction.



Investigations

1. **Chest X-ray & Screen**
 - Widening of superior mediastinum.
2. **Echocardiography**
3. **CT scan & MRI**
4. **Aortography**: diagnostic
5. **Investigations for the cause**: e.g. serological tests for syphilis.

Treatment

Medical treatment:

- **Analgesia**.
- IV infusion of **Na nitroprusside** to reduce the systolic BP to **100 - 120** mm Hg.
- **Propranolol** to keep the heart rate in the range of 60 / min.

Surgical treatment:

Resection of the affected portion of aorta & replacement with a prosthetic graft.

N.B.

- Thrombolysis will kill in this condition, so always look for mediastinal widening in the chest X ray before thrombolysing.

Cardiogenic shock

Definition

Decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume.

Haemodynamic criteria for cardiogenic shock are

1. Sustained hypotension (systolic blood pressure $< 90 \text{ mm Hg}$ for at least **30 min**).
2. A reduced cardiac index ($< 2.2 \text{ L / min / m}^2$).
3. Elevated pulmonary capillary occlusion pressure ($> 15 \text{ mm Hg}$).

Causes

A. Cardiac causes (causes of rapid reduction of COP)

1. Pump failure: **Causes of LVF**.
2. OUT flow obstruction: as **sever AS** or **S. hypertension**.
3. Valvular dysfunction e.g. **acute mitral regurgitation**.
4. Cardiac arrhythmias **Ventricular tachyarrhythmias**.

B. Extra-cardiac causes

1. Global hypoxemia.
2. Myocardial depressant drugs (e.g., B-blockers, Ca blockers).
3. Respiratory acidosis.
4. Pulmonary vascular disease (e.g., pulmonary hypertension)
5. Acute respiratory distress syndrome.

Clinical presentation

Symptoms Low CO.

Signs

General signs

- Cyanotic, cool skin and mottled extremities.
- Pluses: rapid and faint.
- Jugular venous distention.

Local signs

- Heart sounds usually distant, and both third and fourth heart sounds may be present.
- A systolic murmur is generally heard in patients with acute MR or VS rupture.

Investigations as MI (see page 42)

Treatment

- **Vasopressors / inotropic agents**: (Dopamine – Dobutamine).
- **Vasodilators** : as, Nitroglycerin.
- **Analgesics** Morphine sulfate.
- **Diuretics**: as, Frusemide.

Sudden Cardiac Death

Definition

Sudden cardiac death is unexpected natural death from cardiac causes within less than two weeks of recent illness.

Causes

1. Myocardial infarction & ischaemia.
2. Ventricular tachyarrhythmias especially VF.
3. Heart block: Mobitz type II & 3rd grade (**Adam's stock attack**).
4. AS & hypertrophic cardiomyopathy.
5. Fallot's tetralogy.
6. Mechanical causes:
 - Obstruction of blood flow: massive pulmonary embolism ball & valve embolus.
 - Cardiac tamponade.
 - Rupture of heart or aorta.

Clinical presentation

1. **Cardiac**: absent pulse & heart sounds.
2. **Chest**: absent respiration & cyanosis.
3. **C.N.S.**: loss of consciousness, convulsions, brain damage & death.

Treatment

1. Cardiopulmonary resuscitation (CPR):

- A. **A**irway: Removal of foreign materials-Suction - Hyperextension of the neck.
- B. **B**reathing: Mouth to mouth breathing – Mask - Intubations and ventilation.
- C. **C**irculation: Cardiac massage.

2. Further dealing according to ECG pattern:

A. Ventricular tachyarrhythmia VF:

- Defibrillation (300-400 joules) → if no response lidocaine → if no response defibrillation → if no response bretylium → if no response defibrillation.
- (**Response**: VF changes to V. tac) then treatment of V. tac should be admitted.

B. Asystole:

- **Adrenaline** 5-10ml (1 : 10,000) intracardiac or IV (every 5 min).

C. Heart block or severe bradycardia:

- **Atropine** 0.5mg IV (every 10 min up to 5 mg)
- **Isoprenaline** 2-20 mcg/min IV infusion.
- **Cardiac pacing**.

D. Electromechanical dissociation:

- **Intracardiac calcium** 5 ml 10% solution (repeated every 10 min).
- Correction of **acid-base** and **electrolyte**.

3. Treatment of the cause. E.g. Ischemia.